EFFECT OF JAMAN FRUIT AND JAMAN SEED ON BLOOD GLUCOSE AND LIPID PROFILE IN DIABETIC INDIVIDUALS

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IN
HUMAN NUTRITION

BY

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PESHAWAR-PAKISTAN
DECEMBER, 2003
IN THE NAME OF ALLAH
THE MOST MERCIFUL
THE MOST GRACIOUS
AND HE IS THE HELPER
Dedicated To

My Wife

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(SALEEM KHAN)
EFFECT OF JAMAN FRUIT AND JAMAN SEED ON BLOOD GLUCOSE AND LIPID PROFILE IN DIABETIC INDIVIDUALS

Saleem Khan And Alam Khan

ABSTRACT

The effect of jaman fruit and seed on blood glucose, triglyceride (TGL) and total, low-density lipoprotein (LDL) & high-density lipoprotein (HDL) cholesterol was studied in Type-II diabetic individuals for 40 days. One hundred eight (54 male & 54 female), diabetic individuals were selected for the study. The selected individuals were 40-65 years of age having blood glucose and triglyceride in the range of 140-250 and 200-250 mg/dl respectively. These individuals were divided into 9 groups. Each group was having 12 (6 male & 6 female) individuals. Group 1, 2 and 3 were given 6, 12 and 18 jaman fruits per individuals/day respectively. Weight of one jaman fruit was 8 g. No placebo was used for the jaman fruit groups. Groups 4, 5, 6 were given 1, 3 and 6 g jaman seed per individuals per day respectively. Group 7, 8 and 9 were given 1, 3 and 6 g placebo (corn flour) per individual/day respectively. The experiment was for 40 days, 30 days for feeding jaman fruit, seed and placebo and 10 days as washing period. Blood sample were taken from all the groups on day 0, 10, 20, 30 and 40. Blood serum was separated and analyzed for glucose, triglyceride (TGL) and total, low-density lipoprotein (LDL) & high-density lipoprotein (HDL) cholesterol. The jaman fruit and seed were analyzed for zinc and chromium. There was a trend of reduction with both jaman fruit and seed in glucose concentration. However, the reduction was not statistically significant. The
jaman fruit slightly reduced the cholesterol concentration but the reduction was not statistically significant. The jaman seed did not affect the blood cholesterol concentration. The jaman fruit and seed did not affect the concentration of TGL, HDL and LDL cholesterol. The zinc content of jaman fruit and seed was 0.13 and 0.068 mg/kg of sample and the chromium content was 6.00 and 14.00 mg/kg sample. According to this study jaman fruit and seed have no significant lowering effect on blood glucose and lipid profile in diabetic Type-II individuals.
I. GENERAL INTRODUCTION

Diabetes is a chronic disease caused by a lack of or resistance to insulin, which is essential for metabolism of glucose in the body. Insulin is secreted by the beta cells of pancreas. Whenever, there is defect in the secretion or function of insulin, diabetes occur. In both cases carbohydrate is not properly metabolized and there is elevated amount of sugar in the blood. The body homoostatic mechanism removes sugar from the blood and sends the extra sugar to the kidney for excretion in the urine. As sugar is not utilized in the body and is excreted in the urine, the diabetic individuals feel more hunger, drink more water and urinate more often. Diabetes if not controlled, damages nerves and blood vessels, leading to complications such as blindness, heart and kidney disease, nerve problems, gum infections, and amputation. Preventing or delaying the development of diabetes is far better than preventing or delaying the complications of diabetes.

There are four types of diabetes. These are Type-I, Type-II, gestational diabetes and other specific types. Type-I diabetes, is usually diagnosed in children, teenagers, or young adults. In this form of diabetes, the beta cells of the pancreas no longer make insulin because the body's immune system has attacked and destroyed them. Genetic and environmental factors also are involved in the development of this type of diabetes. Type-I diabetes account for about 5 to 10 percent of all diagnosed cases of diabetes. Treatment of this type includes insulin injection and intake calculated amount of food.
Type-II diabetes, is the most common and can be developed at any age usually it occurs at the age of 40 or above. In this type the pancreas does not make enough insulin or the insulin is not working properly. Type-II diabetes account for about 90 to 95 percent of all diagnosed cases of diabetes. Treatment includes drug therapy, diet and natural products therapies and regular exercise.

Gestational diabetes develops during the late stages of pregnancy. Although this form of diabetes usually goes away after the baby is born, a woman who has had it is more likely to develop Type-II diabetes later in life. The hormones of pregnancy or a shortage of insulin causes gestational diabetes. This type develops in 2 to 5 percent of all pregnancies. Specific types of diabetes result from specific genetic syndromes, surgery, drugs, malnutrition, infections, and other illnesses. Such types of diabetes may account for 1 to 2 percent of all diagnosed cases of diabetes.

The new diagnostic criteria for diabetes include a fasting plasma glucose test rather than the previously recommended oral glucose tolerance test. However, in certain clinical circumstances, physicians may still choose to perform the oral glucose tolerance test. A confirmed fasting plasma glucose value of greater than or equal to 126 milligrams/deciliter (mg/dL) indicates a diagnosis of diabetes. Previously, a value of greater than or equal to 140 mg/dL had been required for diagnosis. In the presence of symptoms of diabetes, a confirmed non-fasting plasma glucose value of greater than or equal to 200 mg/dL indicates a diagnosis of diabetes. When a doctor chooses to perform an oral glucose tolerance test (by administering 75 grams of anhydrous glucose dissolved in water, in accordance
with World Health Organization standards, and then measuring the plasma glucose concentration 2 hours later) a confirmed glucose value of greater than or equal to 200 mg/dL indicates a diagnosis of diabetes. In pregnant women, different requirements are used to identify the presence of gestational diabetes.

Type-II is the most common type of diabetes and is the major health problem in the world as well as Pakistan. As the disease is constantly increasing, there is an urgent need to control the disease. Diet is the priority choice to control Type-II diabetes though use of hypoglycemic drugs are common. Proper dietary intake can prevent the incidence of the disease and even can reduce the severity of existing cases. The broad aims of dietary prescription for people with Type-II diabetes remain, first, to abolish the primary symptoms, secondly to minimize the risks of hypoglycemia and thirdly to minimize the long-term macro-vascular and micro-vascular complications which together result in morbidity and shortened lifespan with all types of diabetes. Precaution in eating needs a very strong will and many people may not restrict themselves to a particular way of eating.

Plant products like fruits, seed, and spices are becoming popular in the treatment of Type-II diabetes. Recently Khan and Anderson (2003) have shown that jaman fruit and seed have an insulin potentiating factor which enhances carbohydrate metabolism in rats. However, there is a general view that the results of animal studies may not be applied to humans. Therefore, this study was designed to see the effect of various doses of jaman fruit and seed on blood glucose and lipid profile in Type-II diabetic individuals.
1.1 AIMS AND OBJECTIVES OF THE PROJECT

The overall objectives of the project is to minimize the incidence of diabetes and to cure the diabetes with plant products (jaman fruit and seed). The specific objectives of the project are:

a. To determine the effect of jaman fruit and jaman seed on glucose level in Type-II, diabetic individuals.

b. To determine the effect of jaman fruit and jaman seed on blood lipid profile (triglycerides, total cholesterol, high density and low-density lipoprotein cholesterol in Type-II, diabetic individuals.

c. To determine the effective dosage of jaman fruit/jaman seed for the treatment of Type-II, diabetic individuals.

d. To determine the chromium and zinc content of Jaman fruit and seed and to relate it to the hypoglycemic effect of jaman
2. REVIEW OF LITERATURE

Diabetes commonly known as high levels of sugar in the blood. The excessive sugar then spills into urine. The main reason for diabetes is the high intake of simple sugar in the diet. It is a serious health problem all over the world including Pakistan. The prevalence rate of the disease varies from country to country. According to McCarty and Zinnett, (1997) the worldwide prevalence of diabetes mellitus is expected to double between 1994 and 2010 to more than 240 million people.

In Pakistan, diabetes occurs relatively in younger age, as compared to the western world where it occurs above the age of 40. The onset of diabetes in the country however, occurs in 15-18 per cent of people under 40 and the prevalence of rate of diabetes and IGT increase with advancing of age in both sexes reaching a peak at 55-64 years in men and 65-75 in women. About 25 to 43 percent of people with diabetes (Type-II) have a positive family history (Shera et al, 1994). According to WHO (World Health Organization) reports published in 1995, on the bases of prevalence of diabetes, Pakistan was ranked at number 8 in the world. The health experts suggested that if proper strategies for the prevention of the diabetes were not adopted, Pakistan will be ranked at number 4 in the New year 2025 having 14.5 million people with diabetes (Shera et al, 1994). The unchecked use of steroids by quacks in the rural areas of Islamabad Capital Territory (ICT), contributes diabetes more than 12% and about 40,000 people in the rural areas of Islamabad are suffering from diabetes. The national average for
prevalence of diabetes in the rest of the country was seven to 15% (Zafar 2001). In Karachi, a hospital reports the registration of 796 diabetic patients of which 448 (56%) were male and 348 (44%) were female (Vohra et al 1998). A survey of Bahawalpur city, showed that diabetes mellitus prevails 5.3% in local inhabitants (Shaukat et al 1988). According to Khan et al, (1993) diabetes prevails 1.49% in NWFP.

2.1 TYPES OF DIABETES

There are two major forms of diabetes mellitus, type-I and type-II. Type-I is also called juvenile on-set diabetes mellitus or insulin dependent diabetes mellitus (IDDM). It prevails around 10% of the total diabetic patients. It is more severe, and is caused by insufficient production of insulin and resulting in the abnormal metabolism of carbohydrate, fat and proteins. It is characterized by increased levels of glucose in the blood and urine, acidosis and wasting along with excessive thirst and urination (Vuksan et al 2000).

Type-II is also called adult-onset or non-insulin dependent diabetes (NIDDM), caused 90% diabetic problem. However, most diabetics are Type-II, characterized by hyperinsulinemia or insulin resistance. Roughly 75% of Type-II diabetics have elevated levels of insulin but this insulin is not totally effective (Anderson 1995). It is highly preventable and more benign form of the disease, and if left untreated it can cause severe damage to the cardiovascular and nervous systems. It has no physical symptom and could be detected by glucose intolerance test (Alan and Rubin 1999). Generally, diet alone or diet with drugs are used to
control this disease. In advance stage, insulin treatment is required. Obesity is one of the factor that results in type-2 and contributes to insulin resistance (Khan, et al. 1993) and (Vuksan, et al. 2000).

2.2 ETIOLOGY OF DIABETES MELLITUS

There are several causes of diabetes, some of which are reviewed below.

According to Leslie and Ellior (1994) viral infection i.e. (mumps, hepatitis, mononucleosis, congenital rubella and coxsackie) can infect pancreatic beta cells that finally results in severe diabetes. Whereas, Shahida et al (1995) and Surwit (1993) independently reported that stress cause extreme hormonal fluctuations in the human body and develop either a hyperglycemic or hypoglycemic response that damage insulin producing cell. Other studies reported that nitrates generally found in water, vegetables, preserved meats and fish, are reduced to nitrosamines in the gastrointestinal tract. These generate free radicals and cause damage the insulin-producing beta cells in the pancreas and cause in IDDM (Helgason and Johasson, 1981). Ghittingham (1979) reported that 75% beta cell destruction was caused by autoimmune antibodies in IDDM as compared with 0.5 to 2.0% of non-diabetics. N-nitroso compounds found in smoked and cured meats cause cancer in susceptible individuals producing beta cell damage. Kissebah et al (1995) stated that obesity is associated with a high incidence of NIDDM, cardiovascular disease, hypertension and premature mortality. They reported that obese individuals have a decrease ability to utilized glucose in peripheral tissue or extra insulin during its first portal passage. They
also increased circulating free fatty acids (which reduce glucose metabolism) and
decrease insulin receptor number.

Diet high in simple carbohydrate and fat usually result in the development
of diabetes in the later stages of life. Development of Type-II diabetes is directly
related to diet (Khan and Ahmad, 1994). Marshall et al., (1991) have reported that
high levels of saturated fat and low levels of carbohydrates increase the risk of
developing Type-II diabetes. They further reported that free radical damage,
fasting, diets high in fat, protein and sugar and low in fiber as well as irregular
pattern of eating, exercising and sleeping are the other causes of diabetes.

Whereas, Gottlieb et al (1968) and Kobberling (1971) reported that genetic
factors are involved in both types of diabetes. They reported that 20-50% chance
of IDDM in identical twins while in non-identical twins 5% chance of developing
diabetes. Further they stated that if an identical twin has NIDDM, there is nearly a
100% chance that the other will develop it, but if the twin is non-identical there is
only a 10% chance. Another study revealed that 25% of non-identical brother and
sister of diabetics might develop either type-I or type-II.

Roughly 75% of Type-II, diabetic individuals have elevated levels of
insulin but this insulin is ineffective due to membrane abnormalities (Anderson

Milk contains bovine albumin peptide which trigger an autoimmune
response. It is composed of 17 amino acid sequence identical to a the sequence of
beta cells of the pancreas. When a person develops an allergic reaction to bovine
albumin peptide the body develops antibodies to the 17 amino acid sequence and recognizes the pancreatic beta cells as the same. The body will destroy its own pancreatic cells as a result of this immune recognition (Dosch 1993).

2.3 TREATMENT OF DIABETES

Drugs and diet therapies are the major methods used in the treatment and control of diabetes mellitus. The initial treatments include diet therapy and oral hypoglycemic drugs. In some cases where hypoglycemic agents do not work, the diabetic individuals are placed on insulin therapy. The insulin therapy is successful in the beginning, but with passage of time, the insulin requirement increases and high amount of insulin circulation in the blood is dangerous and promotes other health problems like atheroclerosis and diabetic complication. Drug therapy, whether using hypoglycemic agents or insulin is costly and keeps the diabetic individuals under constant medical supervision and the impression of being sick.

Diet therapy method is economical and feasible in developing countries. People with diabetes consider the restriction on intake of certain foods as precautionary measure for the control of the disease. Diet therapy in diabetic consists basically of precautions concerning diet composition, the amount, distribution and timing of food intake (Carrerini-Davalos, 1987).

The main focus in this method is on the adjustment of macronutrient particularly the content of carbohydrates and fats and restrictions on simple sugars in the diet. Carbohydrate and fats are important in the etiology and control of diabetes (Khan et al, 1993)
The use of herbal products for the treatment of diabetes is growing rapidly because of several reasons. This way of treatment of diabetes has been in used since long (Bhandari and Grove, 1999).

A number of medicinal/culinary herbs have been reported to yield hypoglycemic effect in subject with diabetes (Khan et al., 1990). These include bitter melon, Momordica charantia (Srivastava et al., 1993) Rahman and Lau, 1996); gurnar, gymnema sylvestre (Basakaran et al., 1990; Shanmugasundaram et al., 1990; Bishayee and Chatterjee, 1994); Korean ginsing, Panax ginsing (Sotaniemi et al., 1995); onion, Allium cepa and garlic, Allium sativum (Kochand Lawson, 1996); holy basil, Ocimum sanctum (Rai et al., 1997); flaxseed meal, Linum usitatissium (Cunnane et al., 1993) and Jaman (Ashok et al. 2001; Dewan, 2000).

Spices and other natural products may be hazardous for health and hence must be used with caution. Marles and Fransworth (1994) cautioned that one - two third of the 1123 plants that affect blood glucose may be dangerous, and many of the phytochemicals are hypoglycemic due to metabolic or hepatic toxicity. However, people around the world have long utilized medicinal plants for diabetes safely and with reasonable success (Bailey and Day, 1989; Ivorra et al., 1989; Marles & Fransworth, 1994 and Duke et al., 1998). Botanical products can improve glucose metabolism and over all condition of persons with diabetes not only by hypoglycemic effect but also by improving lipid metabolism, antioxidant status, and capillary function (Brodbyrst 1997).
The use of jaman fruits and seed is traditionally used for the control of diabetes in most clinical practices of the Hakims in this country and elsewhere (Khan et al. 1999). In the contemporary literature there is no evidence of health hazard associated with the consumption of jaman fruit and seed.

Keeping in view the importance of diabetes and the use of jaman for the controlling of diabetes, a research project was designed to evaluate the role of jaman fruit/Seed in diabetes. Previous work on jaman for the purpose is discussed below.

2.4 INTRODUCTION TO JAMAN

The Jaman (Syzygium cumini) or (Eugenia jambolana), belongs to family Myrtaceae. It is locally known as jaman, jammun, java plum, black plum, jambul, Indian blackberry and Rose apple (Zaman and Shariq, 1995). It is widely grown in warm countries such as Pakistan, Sri Lanka, Malaya, Australia, and tropical America. Its fruits are delicious and have a great importance in folk medicine (Chopera, 1956). All the parts of the jaman plants such as barks, fruits and seeds posses medicinal and therapeutic values (Kirtikar et al 1990).

2.5 PRINCIPAL CONSTITUENTS OF JAMAN

Jaman seeds contain certain alkaloids such as Jambosine, glycoside, antimellin which halts the conversion of starch into sugar. Its seeds also contained a phenotic substance called ellagic acid, a trace of pale yellow essential oil, chlorophyll, fat, resin, gallic acid, albumen, etc. The seeds extracts has lowered
The juice of jaman fruit is used for enlarged spleen, chronic diarrhea and urine retention. Water diluted juice as a gargle for sore throat and as a lotion for ringworm of the scalp (Morton and Miami, 1987). The fresh seeds are most effective in diabetes as they quickly reduce sugar in urine (Ashok and Daradka 2001; Zaman and Shariq, 1995). It has been reported that glycoside jamboline stops the pathological conversion of starch into simple glucose and therefore prevent the spell of sugar in the urine. It decreases the quantity of sugar in urine and reduce the unquenchable thirst in diabetes (Morton and Miami, 1987). The seed and bark have several applications in Ayurveda, Unani and Chinese system of medicine. The seeds and barks are used in tropical medicine (Morton and Miami, 1987). So far, no significant side effects have been reported on the use of jaman in controlling diabetes.

2.7 ROLE OF JAMAN IN BLOOD GLUCOSE

Anita et al (2002) administered bittergourd (karela), jaman, and fenugreek seed (methi seeds), in the form of capsule and salty biscuits to different individuals. They reported that fasting and post prandial glucose level of the individuals were reduced. Furthermore, they observed a significant improvement in the serum lipid profile by lowering total, I.DL cholesterol, VLDL cholesterol and triglycerides and by increasing HDL cholesterol level.

Ashok et al (2001) reported the hypoglycemic response of Jaman seeds extract (50% ETOH) (Syzygium Cumini) on diabetic mice. Jaman dosage decreased 37.17% blood glucose just after 3 hours of intake with 6 hours intake
the blood glucose further decrease by 46.68%. Similarly, the blood glucose levels of the alloxan induced diabetic animals were also depleted after 3 hrs and 6 hrs by 46.12 and 65.71% respectively.

Virmani et al (2001) tested a dosage of 10 gm of Jaman seed powder to 10 diabetic individuals for 4 weeks. This dosage decreased the fasting blood glucose 6.99%. They further investigated the effect of jaman dosages after meal, the blood sugar levels reduced 10.56%. The jaman dosages increased cholesterol levels 9.53%, and reduced triglycerides levels 10.35% whereas it slightly increased HDL levels.

Dewan (2000) suggested a dose of 2-3 gms a day jaman seed powdered for the diabetic patients. The jaman seed contained a glycoside which prevents the conversion of starch into sugar.

Teixeira et al (2000) served tea prepared from jaman leaves to diabetic individuals. They found that jaman leaves did not show antihyperglycaemic effect. They also prepared a dose of crude extracts from leaves of Jaman and administered for 2 weeks, to the normal rats and rats with streptozotocin-induced diabetes mellitus to know its effect on post-prandial blood glucose level both treatment gave no anti-hyperglycaemic response.

Ahmed et al (1995) conformed the hypoglycemic action of jaman extract in animal and reported that it reduced glucose level from $161 \pm 2.8$ mg/ml to $134 \pm 3.0$ mg/dl.
tested on normal and hyper-glycaemic adult rabbits of both sexes and two diabetic volunteers (40-50) years. 20 and 40 mg/kg Jaman ethanol extract gave significant results. Whereas, a dose of 40 mg/kg body weight of normal rabbits gave insignificant at the rate of 40 and 80 mg/kg, chloroform extract and 40 mg/kg aqueous extract.

Dietrich (1974) tested aqueous extracts of fresh Jaman seeds (Syzygium cumini) on diabetic rabbits. A single dose of the extract reduced fasting blood sugar (15 to 35%) in four to five hours. He also reported that Jaman contained triterpene acids, oleanolic acid and erategelic acid.

2.8 ADMINISTRATION OF JAMAN DOSAGES

For the prevention diseases, the recommended doses of the medicine play a vital role. Prince et al., (1998) have recommended a dose of 2.5 , 5.0 g/kg and 7.5 g/kg body weight of aqueous extract of jaman seed for the treatment of diabetes. Another report suggests a dose of 2-3 gms a day jaman seed powdered for the diabetic patients by (Dewan, 2000). Morton and Miami, (1987) used Jaman fruit and seed 2 to 3 times per day for diabetic mellitus or glycosuiria. Another report suggested that the fluid extract 1/2 ounce should be taken in 8 oz. hot water 1 hour before breakfast and before going to bed (Anonymous, 2002).

Proximate Composition of Jaman Fruit And Seed

Jaman fruit contains sufficient amount of sodium 391.5 mg, potassium 278.4 mg, calcium 116.0 mg, iron 0.58 mg, zinc 0.29 mg and manganese 0.58 mg. In addition it contains glucose, mannose, sucrose, alanine, arginine,
asparagine, tyrosine, glutamine and cysteine Noomrio (1996). According to another report jaman fruit contains 83.7-85.8 g moisture, 0.7-0.129 g protein, 0.15-0.3 g fat, 0.3-0.9 g crude fiber, 14.0 g carbohydrate, 0.32-0.4 g ash, 8.3-15 mg calcium, 35 mg Magnesium, 15-16.2 phosphrous, 1.2-1.62 mg Iron 26.2 mg sodium, 55m Potassium, 0.23 mg copper, 13 mg sulfer, 8 mg chlorine, 80 I.U. Vitamin A, 0.008-0.01 mg Thiamine, 0.009-0.01 mg Riboflavin, 0.2-0.29 mg Niacin, 5.7-18 mg Ascorbic acid, 7 mg Choline, 3 mcg Folic acid per 100 g of edible portion.

Venkitakrishnan et al (1997) analysed jaman fruits at ripening, ripe and edible stage. They reported that chlorophylls, carotenoids, free phenols and flavonols decreased but anthocyanins increased during ripening. Starch and non-reducing sugar levels also decreased but sugar content increased with increases in reducing sugars.

2.9 ROLE OF TRADITIONAL MEDICINES (OTHER HERBS) IN DIABETES

Alam and Mahpara (2003) stated that diabetes has a global health problem. It affects carbohydrate metabolism and cause high blood glucose and glycosuria due to dysfunction of pancreatic beta cells and insulin resistance. Further, in advance stages also altered protein and lipid metabolism. Its contributing factors are heredity, age, obesity, diet, sex, sedentary life style, socio economic status, hypertension and stresses. They reported that various therapies i.e. drug, diet and spices are used to control blood sugar. Presently, the spices
trend is growing among the diabetic individuals. Such as cinnamon, cloves, bay leaves and turmeric have an insulin potentiating activity. Further they reported that cinnamon contained methyl hydroxy chalcone polymers which reduce blood sugar in diabetic individuals.

Okyar et al (2001) evaluated Aloe vera [A. barbadensis] leaf pulp in diabetic and non-diabetic rats. They reported that A. vera leaf pulp and gel extracts were ineffective to reduce blood sugar of non-diabetic rats but the extract were found effective in diabetes.

Mani et al (2000) provided a dose of 2 g/day spirulina tablet to the diabetic individuals for 2 months. They reported a significant reduction in blood sugar, glycated serum protein, triglycerides, cholesterol, free fatty acid, LDL-c, VLDL-c and HDL-c/LDL-c ratio.

Jeppersen et al (2000) reported that natural sweetener stevioside reduce blood glucose in diabetic patients. They fed such plants to mouse and found that stevioside enhanced insulin secretion. They suggested that such sweeteners could be used to enhance insulin secretion in diabetic individuals.

Broadhurst et al (2000) evaluated the possible effects of 49 herb, spice and medicinal plants extracts on insulin function in the insulin dependent utilization of glucose using a rat epididymal adipocyte assay. Cinnamon was the most bioactive product followed by witch hazel, green and black teas, allspice, bay leaves, nutmeg, cloves, mushroom and brewer's yeast. The glucose oxidation enhancing bioactivity was lost from cinnamon, tea, witch hazel, cloves bay leaf
Ramirez (1999) evaluated the effect of ethanol-aqueous extract of *Curcuma longa* in atherosclerotic rabbits. They orally administered 1.66 and 3.2 mg/kg body weight of turmeric extract and they noted that low dose significantly decreased the susceptibility of LDL to lipid peroxidation and lower dose have lower levels of cholesterol, phospholipids and triglycerides in LDL as compared to higher dose 3.2.

Purohit et al (1999) tested *Curcuma longa* extract (50% EtOH) in hyperlipidaemic rabbits. They reported that the serum cholesterol of rabbits dropped from $940.7 \pm 50.10$ and $119.2 \pm 21.27$, 75.55 % and 87.32 %. Similarly, phospholipids and triglyceride levels were also reduced. The tissues lipids profiles of liver and heart muscles showed similar changes.

Akhtar and Akhtar (1999) studied the effect of various doses of *Pongamia pinnata* flowers in normal and diabetic individuals. They administered a dose of 5 g powdered flowers with 200 ml of tap water to normal individuals. While group 2-4 received 3, 4 and 5 g of powdered flowers and their blood glucose levels were determined at 1, 2 and 3-h intervals. They observed a significant reduction in blood glucose levels of normal and diabetic individuals treated with powder. The mean reduction of blood glucose levels in normal individuals was noted 22.5±2.3%. While reduction in blood glucose levels of diabetic individuals of dose 3 g, 4 g, and 5 g were observed 31.1±2.1%, 35.8±2.8%, and 47.2±3.8% respectively.

Ahmad et al (1999) tested *Karolla* (Momordica charantia) on fasting and post prandial (2 hours after 75 gm oral glucose intake) serum glucose levels in
100 moderate diabetic individuals. A significant reduction in fasting and post-prandial serum glucose levels was observed in 86 individuals and 5 individuals showed lower fasting serum glucose.

Mahdi (1998) administered a diet-contained barley to the adult diabetic rats. This diet showed a positive effect on blood glucose when compared with starch or sucrose diet. He found that this beneficial effect of barley might be due to chromium 5.69 micrograms/g.

Khan et al. (1998) studied the influence of spices- Murraya Koenigii and Brassica juncea on rats fed atherogenic diet. A high fat cholesterol diet containing 10% Murraya Koenigii leaf (curry leaf) or 10% Brassica juncea (mustard) seeds or a high fat cholesterol diet alone was fed to Sprague-Dawley rats 3 months (n=12 per diet). The plasma lipoprotein profile at 3 months showed that these spices reduced the concentrations of cholesterol, TGL and phospholipids in serum, aorta, liver and heart (P<less 0.01). The low-density lipoproteins and very low-density lipoprotein fractions were also decreased and the HDL fraction was increased. It is suggested that these 2 common spices could play a significant role in controlling the development of hypercholesterolaemia and arteriosclerosis.

Chithra and Leelamma (1997) reported that hypolipidemic effect of coriander seeds (Coriandrum sativum) and its mechanism of action. Female albino Sprague-Dawley rats (65-70g) were randomly assigned to receive a high fat diet (rodent chow + coconut oil (15%) + cholesterol (2%) or a high fat diet including coriander seeds (Coriandrum sativum), 10%) for 75 days. The levels of
cholesterol decreased in the tissue of rats fed coriander seeds (89.10±2.60, 1050±30.50 and 90.43±2.62 vs. 160.30±4.65 mg/100 ml, 1751.20±50.80 and 281.60±8.17 mh/100g in serum, liver and heart, respectively). Corresponding values for triacylglycerol were 7.390±0.214, 329.75±9.56 and 26.93±0.78 vs 14.80±0.43 mg/100ml, 797.90±23.14 and 71.80±2.10-mg/100 g, respectively. Beta-Hydroxy, beta-methyl glutaryl CoA reductase (hydroxymethylglutaryl-CoA reductase (NADPH)-phosphates) and plasma lecithin-cholesterol acyltransferase (phosphatidylcholine-sterol O-acyltransferase) activity increased and HDL cholesterol increased in the experimental group vs. the control group. They concluded that the hypocholesterolaemic effect of coriander seeds is as a result of increased activity of plasma LCAT enhanced hepatic bile acid synthesis and the increased degradation of cholesterol to fecal bile acids and neutral sterols.

Storlien et al (1996) and Eritsland et al (1994) have been collected muscle biopsies from individuals during surgery and reported that all the sample have higher degree of membrane saturation and more insulin resistance was observed in the individuals.

Storlien et al (1996) and Pan et al (1995) stated that a diet contains higher SFA, hydrogenated fat and n-6 PUFA, significantly affect insulin efficiency and glucose response. Further, they stated that skeletal muscle is the major site of insulin-stimulated glucose utilization. Due to use of higher amount of fat increase the accumulation of storage triglyceride in skeletal muscle (i.e., marbling), which leads directly to insulin resistance in the individual muscles.
Sharma et al (1996) tested 25 g fenugreek seeds (Trigonella foenum-graecum) in diabetic individuals for 24 weeks. They divided 25 g fenugreek seed in 2 equal doses and added to the diet in lunch and dinner. A significant reductions in cholesterol, LDL, VLDL cholesterol and triglycerides was observed.

Khan et al (1996) studied the biochemical response in rats to the additions of curry leaf (Murraya koenigii) and mustard seeds (Brassica Juice) to the diet. Three groups of 12 weanling male albino rats were fed for 90 days on 1 to 3 diets: a standard laboratory rat diet supplemented with 20% coconut oil (high fat control diet) of the high fat diet supplemented with 10% curry leaf (M. koenigii) or 10% mustard seeds (B. Juicea). Feed was offered at a level of 10% body weight. At the end of the trial total serum cholesterol, HDL, LDL and VLDL fractions, release of lipoprotein into circulation, lecithin cholesterol acyl transferase (LCAT) activity and lipoprotein lipase activity were examined. Curry leaf and mustard seeds decreased total serum cholesterol and LDL and VLDL, increased HDL, decreased the release of lipoprotein into the circulation and increased LCAT activity.

Sotaniemi et al (1995) narrated that Korean ginseng (Panax ginseng), 200 mg/day was administered to diabetic patients for 8 weeks and found that ginseng improved their mood, physical activity and lowered fasting blood glucose, glycosylated hemoglobin and body weight.

Kissebah and Hennes (1995) reported that obesity caused diabetes, cardiovascular diseases, hypertension and premature mortality. In obese
individuals glucose utilization is become low in peripheral tissue. The circulating free fatty acid is high and limited receptor number.

Gomes (1995) reported that extract of black tea significantly reduced the blood glucose level and posses preventive and curative effect in diabetic animals. He also reported that green tea and black tea have anti-diabetic activity.

Rahuram et al, (1994) and Sharma and Rahuram (1990) observed the hypoglycemic effect of fenugreek seeds in 15 NIDDM subjects. Incorporation of fenugreek in diet produced a significant fall in fasting blood glucose and improvement in glucose tolerance, by improving peripheral glucose utilization.

Gupta et al (1994) provided 3.5g psyllium husk to the diabetic individuals for 90 days with hyperlipaemia. They reported that psyllium husk significantly decrease total cholesterol 19.7%, LDL cholesterol 23.7%, triglyceride 27.2%, and the ratio of LDL-C to HDL-C 24.1%.

Dutta (1994) and Hauge (1988) stated that abnormal cell membrane phospholipid profile has a major significance in both types of diabetes. The insulin stimulates and glycogen inhibits the desaturase enzyme system and also incorporates PUFA in cell membrane. The fatty acid composition of membrane lipids affects the action of insulin. Further, a data have shown that an increase in membrane fluidity by using higher dietary PUFA increases the number of insulin receptors and insulin actions. In diabetes and in chemically induced diabetic animals models, insulin administration and LC-PUFA supplementation (i.e. evening primrose and fish oils) significantly improve the membrane abnormalities.
Bhardwaj et al (1994) tested a herbal powder that contains guar gum (Cyanopsis tetragonoloba), methi (Trigonella foenum-graecum [T. foenum-graecum]), tundika (Cephalaria indica [Coccinia grandis]) and meshasringi (Gymnema sylvestre) in 30 control and 30 diabetic individuals for a month. They reported a significant reduction in low density lipoprotein cholesterol after treatment. A side effects like mild flatulence and looseness of bowel in 40% of the cases was recorded.

Soni (1992) provided 500 mg of curcumin per day for 7 days to healthy volunteers and reported that reduced serum cholesterol 11.63%, serum triglyceride 33%, and HDL Cholesterol 29%.

Akhtar et al (1992) hypoglycaemic effects of Withania Coagulans and Zizyphus Sativa was studied in normal rabbits and diabetic individuals. No hypoglycaemic effect was found in aqueous extracts of Withania coagulans while its ethanolic extracts showed significant results after 24 hours of drug intake. Maximum reduction (35 and 31%) in blood glucose level was found in diabetic individuals after 4 hours of drug ingestion. Zizyphus Sativa chloroform extract produced a highly significant glucose lowering effects as compared to its ethanolic extract. The animals loaded with glucose and the two test drugs showed a highly significant result with in a time period of 1 to 2 and a half hours post drug administration.

Shanmugasundaram et al (1990) reported that Gurmar (Gynnema Sylvester) has the ability to abolish the taste of sugar and is known as the "sugar
destroyer". It contains gymnemic acid which block the sensation of a sweet taste, that comes in contact with the tongue. This herb is best known for its treatment in diabetes and has been used in the treatment of diabetes mellitus in India for over 2000 years. Its properties make it the herb of choice for improving sugar control in diabetic conditions.

Sharma et al (1990) tried 100 g/day fenugreek seed (Trigonella foenum-graecum) powder in 10 diabetic patients and after 10 days their fasting glucose decreased by 30% and glucose tolerance improved in the fenugreek group while sugar excretion dropped 54% and no increase was noted in insulin. They reported that fenugreek seed contain protein, saponins, and the hypoglycemic phytochemicals coumarin, fenugreekine, nicotinic acid, phytic acid, scopoletin, and trigonelline.

Sharma et al (1990) reported that Fenugreek seeds contains alkaloid trigonelline, nicotinic acid and coumarine which help to stabilized blood glucose levels.

Khan et al (1990) reported that some of the spices like Cinnamon, Cloves, Bay Leaves and Turmeric have insulin-potentiating effect in vitro. These spices have been used for taste and flavor development in food preparations. Such spice come from dried aromatic plants or trees and may be the bark, roots, seeds, fruits, buds or the berry of these plants. Presently, the trend to use spices and other natural products is growing to control diabetes. Controlling diabetes with spices is more popular, an appropriate and economical for diabetic individuals in developing countries.
Frati et al (1990) tested 500 g nopal (Opuntia spp.) in cooked, raw and water in diabetic individuals. They reported that after 180 minutes, their fasting glucose was reduced 22-25% by nopal as compared to 6% water. Similarly, a dose of 1mg/kg/day of opuntia fuliginosa extract was given for 8 weeks along with insulin normalized blood glucose to the diabetic rats. After seven weeks the O. fuliginosa extract was given alone and glycemic control was maintained. O. fuliginosa, but not insulin, also normalized glycosylated hemoglobin levels but this effect may be due to fiber alone.

Edelberg (1990) a clinical trial have been conducted on Fenugreek seed’s and he reported that Fenugreek seed improve blood sugar control in type-2 diabetic individuals. He recommended a dose of 5 to 30 gms Fenugreek seed three times a day for the diabetic individuals. It is rich in fiber and slow the rate of gastric emptying, inhibits the absorption of glucose in the intestine and lowers the cholesterol levels. He noted that in some people this seed caused stomach upset.

Baskaran et al (1990) administered orally 400 mg/day gurmar (Gymnema sylvestre) leaves to the diabetic individuals for 18-20 months. They reported that gurmar extract stimulate insulin secretion, lower cholesterol and triglycerides without side effects. They also observed that the extract was superior than medication for blood glucose stabilization and lowering of triglycerides. Further, they suggest that the supplementation may regenerated/repaird beta cell in diabetic individuals.

Iyer and Mani (1989) tested bay leaves (Murraya Koenigi) in diabetic individuals. They reported a reduction in fasting and postprandial blood sugar
levels and no significant changes in lipid profile, glycated protein and amino acids.

Jacob et al (1988) probed the effect of Indian gooseberry (amla) in normal and hypercholesterolaemic men aged 35-55 years for 28 days in raw form. They reported a significant reduction in cholesterol levels of normal and hypercholesterolaemic individuals. When the supplement was stopped their serum cholesterol levels of the hypercholesterolaemic individuals return to the initial levels.

Thakur (1985) stated that emblica officinalis significantly reduced serum cholesterol, aortic cholesterol and hepatic cholesterol in rabbits. They found that emblica officinalis did not influence euglobulin clot lysis time and platelet adhesiveness or serum triglyceride levels.

Khare et al (1983) tested gurmar 10 g/100 ml an aqueous decoction powdered leaves on normal and diabetic patients for 15 days. They administered a dose of 2 g TDS to the normal and diabetic patients and significant hyperglycemic reduction of 32% was noted.

Kedar and Chakraborty (1981) evaluated the effect of Gurmar and other herbal plants prepared into Gurmar preparation which is a combination of leaves of gurmar 100 g, bael 100 g, Neem 200 g and seeds of Java plum 25 g. They used 50 ml of aqueous extract of gurmar preparation after lunch and 50 ml after dinner was administered orally for 7 days in 10 diabetic patients ages 40-65 yrs. The found 43% reduction in the level of hyperglycemia.
Arun et al (1981) provided garlic to 20 healthy volunteers for 6 months and they were followed for 2 months without garlic administration. They reported that garlic significantly lowered serum cholesterol and triglycerides but increase the high-density lipoproteins. Further, they select 62 patients having coronary heart disease and elevated serum cholesterol. Such individuals were randomly divided into group one group was fed garlic for 10 months and the other as a control. They reported that garlic decreased serum cholesterol, triglycerides, low-density lipoprotein and increased the high-density protein.

Bordia et al (1979) administered a dose of 2.5 g Fenugreek twice daily for 3 months to diabetic individuals. They observed a significant decrease in blood lipids and cholesterol levels.

Mitra et al (1975) tried 50 mg hydroalcoholic extract from leaves and stems of gurmar (Gynnema sylvestre) in diabetic patients (17 M & 3 F), ages 49-64. They administered a dose of 1 capsule BDS after meals to start with then 1 capsule TDS after meals. They observed a significant reduction in blood sugar after 1 year of medication about 60% of the diabetic individuals were benefited from this treatment and no side-effect or toxicity was observed except mild hypoglycaemia in one case.

2.10 ROLE OF PHYTOCHEMICAL IN DIABETES

Stilling et al (1999) reported that starch free bread showed significant improvement in blood glucose and lipid levels in diabetic individuals. Further they recommended that diabetic individuals eat regular small meals contains carbohydrate and protein in the ratio 2:1 to regulate and maintain glucose levels.
Knekt et al (1999) and Dunta et al (1996) reported that regular exercise of diabetic individuals help to decrease body fat, serum cholesterol and triglyceride. It also improves circulation, self-esteem and self-image and reduces emotional stress. It increases tissue levels of chromium and number of insulin receptors in IDDM to lower blood glucose. Following exercise, additional decrease in blood glucose occurs, as it is stored in muscles as glycogen. However, the power to reduce the blood sugar is destroyed when glucose levels are high at the beginning of exercise, especially if ketones (by products of fat break down) are present. For each 30-45 minutes of exercise, 10-15 grams of extra carbohydrate are needed. Regular exercise for twenty minutes four times each week gives good result.

It is further reported that capillary function was also improved by horse chestnut, billerry, ginkgo, centella asiatica and various standardized anthocyanin preparations (Duke et al 1997).

Broadhurst (1997) reported many of the biologically active phytochemicals in plants, which have bitter or astringent taste. These products have a benefits to control diabetes with out inhibition of glucose absorption, including stimulation insulin secretion or action, improving insulin binding, improving capillary function, and preventing PUFA peroxidation.

It has been reported that phytochemical improve skeletal capillary function that was slower in diabetic individuals. The phytochemical potentiate the action of insulin and they recruit more GLUTE-4, from the cytosole and increase glucose utilization.
Archimowicz et al (1996) and (Morimoto et al 1986) reported that supplementation of buckwheat, butcher's broom and hydroxyethyl rutside have significantly decrease diabetic retionpathy in humans due to improve local circulation in the retina. They also lowered cholesterol and triglyceride and raised HDL cholesterol. Cinnamon also contained procyanidian dimers and oligomers that improve capillary function.

Hallfrisch et al (1995) and Rodriguez et al (1998) reported that whole foods, water soluble fiber i.e. beans, oat bran psyllium husks, pears, apples and vegetables should be included in the diabetic diet which slow down the digestion and absorption of carbohydrate and prevent rapid increase in blood sugar.

Hersey et al (1994) and Helmrich et al (1991) stated that vitamin E prevented blood clotting, protect blood vessels from damage and reduces glycosylation.

Sheela and Augusti (1992) reported that onion and garlic also have a beneficial effect on blood glucose levels.

Daves et al (1992) and Baker et al (1996) stated that diabetic individuals should avoid drink and smoking and adopts good lifestyle. Drink inhibits gluconeogenesis and increases risk of hypoglycaemia in people taking insulin and cause eye and nerve damage. Diabetic individuals and smoker should not drink on an empty stomach and be careful they have a high risk of kidney damage and heart disease.
Gin et al (1991) and Simopoulos (1999) reported that omega-3 fatty acid present in fish that protect against atherosclerotic disease, lower cholesterol and triglyceride levels. Omega-6 present in vegetable oils has been shown to offer protection against the development of diabetic neuropathy. Both of these oils reduce after-meal hypoglycaemia and increase tissue sensitivity to insulin.

Feskens et al (1991) stated that vegetarian and vegan diets reduce the risk of diabetes and low animal protein diets lower kidney damage and improve glucose tolerance.

Zhang (1990) reported that Asian ginseng increase the release of insulin from the pancreas and increases the number of insulin receptor.

Scala et al (1990) reported that vitamin C helps to reduce glycosylation and sorbitol levels.

Khan et al (1990) reported that common spices such as cinnamon, cloves and bay leaves have showed insulin potentiating action in vitro. Most of the biologically active phytochemicals in plants which have bitter or astringent taste. These products have a benefits to control diabetes with out inhibition of glucose absorption, including stimulation insulin secretion or action, improving insulin binding, improving capillary function, and preventing PUFA peroxidation.

Lettle et al (1987) reported that diabetic individuals should not used processed foods, additives, smoked and cured meats preservative, milk fat, cholesterol and sugar, but small amounts of fructose found in fruit do not cause rapid increase in blood sugar level.
Jenkins et al (1981) and Toeller et al (1999) suggested that diabetic individuals generally eat low glycaemic index foods, to stabilize their blood glucose levels. The glycaemic index was developed to measure the rise of blood glucose after eating particular foods. However, the glycaemic index should not be the only dietary guideline and quality whole foods always need to be considered along with some high fat foods such as ice cream and sausage with a low glycaemic index. A diet containing fats contributes to heart disease the leading killer of people with diabetes.

Diabetic individuals need proper management to control blood glucose by use of insulin or drugs. These individuals must get correct nutrition, supplement, exercise, and change the lifestyle good health can be improved. It is reported that diet contains carbohydrate, plant fiber, cereal grains, legumes, roots vegetables and restricts simple sugar and fat intake have a positive metabolic effect in diabetic individuals. The diabetic diet must have 70-75% complex carbohydrates, 15-20% protein and 10-25% fat and 100g fiber per day. The formulated diet not only control blood glucose but also increase tissue sensitivity to insulin, reduces cholesterol, triglyceride and encourages fat loss. Similarly, 16 type-1 individuals were treated with plant fiber diets, their average insulin requirement dropped by 38% Anderson (1977) and Anderson and Ward (1979).

2.11 ROLE OF MINERAL IN DIABETES

Trottier (2000) reported that high chromium yeast supplementation improved glucose tolerance through a decrease in hepatic excretion of insulin.
Diabetes mellitus and cardiovascular diseases cause low dietary intake of chromium. Chromium supplementation improves glucose intolerance in diabetic individuals. In case of severe neuropathy and glucose intolerance of a patient on total parental nutrition, who received recommended levels of chromium increase insulin binding to cells, insulin receptor number and receptor kinase activities leading to explain the mechanism of action of chromium and its role in the prevention and control of diabetes (Anderson et al, 2000).

Humphries et al (1999) studied 179 adult age 30 through a 24-hour dietary interview and their data was analysed. They reported that magnesium intake was found to be correlated with insulin resistance and the results of this study suggest a possible role for dietary magnesium in insulin resistance.

Magnesium is present in higher concentration in living cells. Plasma and intracellular magnesium concentration are regulated by several factors including insulin. It is stated different studies have revealed that insulin regulate the shift of magnesium from extra cellular to intra cellular space ad intracellular magnesium concentration has effective in regulating insulin action, glucose uptake and vascular tone (Paolisso, 1997).

Magnesium is one of the most abundant ions present in living cells. In human subjects the plasma concentration of magnesium remains remarkably constant. Plasma and intracellular magnesium concentrations are regulated by several factors, including insulin. Most of the Studies have demonstrated that insulin may modulate the shift of magnesium from extra cellular to intra cellular
space. Intracellular magnesium concentration has also been shown to be effective on modulating insulin action as well as offsetting the effect of other ions. A low intracellular magnesium concentration as found in non-insulin dependent diabetes mellitus and in hypertensive patients. Both events are responsible for impaired insulin action and a worsening of insulin resistance in non-insulin dependent diabetic and hypertensive patients (Paolisso, 1997).

Deficiencies of essential mineral and specially those involved in the metabolism of carbohydrates i.e. chromium a key constituent of the glucose tolerance factor, is vital to proper blood sugar control. Without chromium, insulin action is blocked and glucose levels are elevated. Other essential minerals are manganese, zinc, potassium and magnesium also important for metabolism. These nutrient are all depleted in refined grains as is three-quarters of the chromium, manganese and zinc tend to be concentrated in the bran and the germ (Anderson 1996).

Anderson et al (1996) reported that chromium store mainly in the kidney and less in amount in the liver, spleen, lungs and heart. Generally plasma chromium is not in equilibrium with tissue store.

Ravina et al (1995) provided 1000 and 200 ug per day chromium picolinate to the diabetic individuals and reported a positive response in higher amounts as compared with 200 ug per day.

Goldfine (1995) reported that vanadyl sulfate therapy significantly decrease insulin requirements and cholesterol levels of both insulin-dependent
diabetes mellitus and non-insulin dependent diabetics. He observed an increase in basal nitrogen-activated protein and S6 kinase activities in mononuclear cells that mimicked the effect of insulin stimulation in controls.

Anderson (1995) reported that chromium deficiency is a causative factor for type-2 diabetes and producing symptoms including fasting hyperglycemia, impaired glucose tolerance decrease insulin binding and receptor number, decrease HDL and increase total cholesterol and triglyceride.

Anderson (1995) reported that chromium tissue stores are depleted by inadequate intake or absorption, pregnancy. Further, depletion caused by strenuous exercise, intake of refine carbohydrates and processed foods, with age, infections and trauma.

Cunningham (1994) reported that zinc should be taken with a dosage as high levels may increase glycosylation.

Rimm et al (1993) stated that chromium helps to lower cholesterol and triglyceride levels and increase insulin sensitivity.

Taylor et al (1992) reported that Western diets provided less chromium and cause long-term depletion of body chromium store. It has been estimated most of the U.S. population does not obtain the safe and adequate daily dietary intake of 50-200 mcg/day chromium. Similar depletion of chromium have been observed in Canada, UK and Finland.

Murray and Pizzorno (1991) reported that chromium is a key constituent
of the "glucose tolerance factor", and normalized blood sugar and its ability to normalized insulin.

Pederson (1989) administered vanadyl sulfate to streptozotocin-induced diabetic rats for three week and reported that stimulates glucose uptake and metabolism to normalized blood glucose. Further they observed that insulin levels were still depressed, glucose tolerance was normalized and slight increase in insulin secretion from pancreas but the islets in the pancreas were almost normalized to control size and insulin content and the rats remained normoglycemic even after 13 weeks of withdrawal.

Kozlovsky et al (1986) and Anderson et al (1990) stated that a diet high in refined grains and sugars aggravate chromium depletion. They reported that these foods contained low amount of chromium but chromium is necessary to metabolize them. Further, consumption of high sugars and insulinogenic foods increase chromium excretion in urine 10-300%.

Ghannam (1986) provided 200 mcg of chromium picolinate to both sexes and reported that improved insulin resistance 62% in women and 50% in men with in 10 days in diabetic individuals. He further reported that it binds with insulin and increase up to 100 times the hormone's main mission of converting glucose into carbon dioxide in test tube experiments.

Potter (1985) administered 200 mcg of chromium chloride daily to 5 elderly patients for 12 weeks and reported that chromium supplementation has improved glucose tolerance.
Anderson (1984) reported that exercise increase the metabolic rate, insulin receptors in the muscle cells that activate and increase glucose utilization. This utilization requires concomitant action by chromium. However, it does not appear that chromium is efficiently recycle by the body. Urinary chromium excretion increases significantly following exercise.

Alam et al (1983) analyzed jaman fruit, jaman seed and reported that jaman fruit contained 1461ng/gm Cr, 8.5ng/gm Cu, 21.4ng/gm Zn, 857ng/gm Fe. While jaman seed contained 119.2ng/gm Cr, 7.7ng/gm Cu, 3.4ng/gm Zn and 73.2 ng/gm Fe.

Nath et al (1979) complemented 400 ug or more chromium as chromium chloride to the diabetic individuals reported a beneficial effect on people who are suffering from diabetes mellitus.

Toepfer et al (1977) reported that the biologically active extract from brewer's yeast contained chromium, nicotinic acid, glycine and glutamic acid. They provided further evidence for their claim by synthesizing biologically active complexes comprised of trivalent chromium, nicotinic acid, glycine, cysteine and glutamic acid. Biologically active chromium is that chromium, which potentiates insulin activity, measured in-vitro.

2.12 ROLE OF VITAMIN IN DIABETES

Romero (1999) narrated that biotin reduce glucose by several different mechanism. It He reported that it is a cofactor of enzymes that needed for fatty
acid synthesis, utilization of glucose and stimulation of glucokinase. Further, biotin has also found to stimulate the secretion of insulin in the pancreas of rats, which also has the effect of lowering blood glucose.

Diabetes increase oxidative stress and cardiovascular complication. Davi (1999) provided 600 mg of synthetic α-tocopherol to the diabetic individuals for 14 days and reported a reduction in oxidative stress. No effect of α-tocopherol on blood glucose was observed.

Zhang et al (1997) reported that biotin deficiency results in impaired utilization of glucose.

Maebashi (1996) tested 9 mg/day, biotin for a month in diabetic individuals and reported that fasting blood glucose levels decrease by an average of 45%.

Mandrup (1993) stated that Niacin helps to restore beta cells, slow down their destruction in the early stages of diabetes and prevent the development of type-I.

Reduction in blood glucose levels was also observed when a dose of 16 mg biotin daily administered to diabetic individuals for a week (Coggeshall, 1985).
3. MATERIALS AND METHODS

3.1 LOCATION OF THE STUDY

This trial was conducted in the Department of Human Nutrition, NWFP Agricultural University Peshawar. The diabetic volunteers were registered with the help of the dispensaries at NWFP, Agricultural University, Peshawar, Islamia College, Peshawar, Pakistan Medical Research Council (PMRC) at Khyber Medical College Peshawar, Hayat Abad Medical Complex (HMC), Peshawar. Basic Health Unite Reagi and Lady Reading Hospital (LRH) Peshawar, Pakistan. The volunteers screened for this trial were residing in the vicinity of Peshawar City.

3.2 SAMPLE SIZE

One hundred and eight Type-II diabetic individuals of both sexes and of age 40 years or above were registered for this study.

Screening of Subjects

Before registration non-insulin diabetic individuals were informed through personal contact/local health worker. These diabetic individuals were called to the department of Human Nutrition on specified dates for screening test. They were instructed to come with fasting state for screening test as well for other information. The screening processes were continued for few weeks. At the time of screening name, address, sex, exercise level, medication and dose of medication were noted in a questionnaire given below. Blood samples were taken
only from those individuals who were in the age range of 40-65 years, who were not on insulin therapy and who are not taking medicine for other diseases. Serum glucose and triglyceride of these individuals were determined.

3.3 CRITERIA FOR SELECTION OF DIABETIC INDIVIDUALS

The criteria for selection of the diabetic individuals was that they should be Type-II diabetic, in the age range of 40-65 years and of both sexes. They should not be on insulin therapy and should not be on any medication other than the diabetes ones. Their fasting serum glucose should be in the range of 140-250 mg/dl and their fasting triglyceride should be in the range of 200-250 mg/dl.

Subject Selection

One hundred and eight (54 male and 54 female) on the above criteria were registered for the study. As sufficient diabetic individuals of the required criteria were not available at one time, so registration of the individual was done at different time. A meeting of the registered individuals was called in the department of Human. The protocol of the study was explained to them. After registration, the individuals were told, "The research is to find a new treatment for diabetes and will continue for 40 days. There will be no restriction to use your own diabetic medicine during the whole trial period. The experimental product (Jaman fruit, Jaman Seed and Placebo) were given to all the selected individuals for 30 days of the experiment and no other product were used except routine diabetic medicine for the next 10 days. Fasting blood sample was also taken, before intervention (day 0) of the experiment and on the 10th, 20th, 30th and after
intervention on day 40 of the experiment. Those who were agree to protocol of the trial, they were given a consent form for signature. After signature, they were the subject of the study.

"I understand the importance of this trial and voluntarily register myself for the experiment and obey the trial protocol."

Name of the Diabetic Individuals.........................

Signature or Thumb sign of the Individuals..................

Address of the Diabetic Individuals........................

3.4 JAMAN FRUIT AND SEED (Natural Plant Product)

Jaman fruit (Eugenia Jambolana), Jaman seed and placebo was purchased from the local market at Peshawar, NWFP.

Grinding of Sample

Jaman fruits were cleaned, washed and then packed in plastic bags according to the specific doses with the request to please keep the fruit in refrigerator. The jaman seed were also cleaned physically to remove stone and other unwanted material. The clean seed were then grinded to get powder, packed and properly labeled.

3.5 PREPARATION OF DOSES FOR JAMAN FRUIT GROUP

Fresh jaman fruit were provided to the selected diabetic individuals. Different doses 6g, 12g and 18g edible portion of jaman fruit that was calculated in terms of numbers of jaman fruits 6no, 12no and 18no. Packages of various
doses of Jaman fruits (6no, 12no, 18no) were prepared. These packages of various doses were distributed among the selected individuals before the feeding trial.

3.6 PREPARATION OF DOSES FOR JAMAN SEED AND PLACEBO GROUPS

The recommended doses of jaman seed and placebo were prepared as follow. Packages for dose 1g/day (0.30g, 0.35g and 0.35g) of jaman seed and placebo were prepared. Similar packages of dose 3g/day (1g, 1g and 1g) and dose 6g/day (2g, 2g and 2g) were also prepared from jaman seed and placebo.

3.7 FEEDING PROTOCOL OF THE TRIAL

The clinical trials were continued for 30 days. The 108 Type-II diabetic individuals were divided into 9 groups. Each group was having 12 individuals (6M and 6F). Groups 1, 2 and 3 were assigned to jaman fruit, groups 4, 5 and 6 were assigned jaman seed and groups 7, 8 and 9 were assigned to placebo. The individuals were allowed to used their routine diabetic medicine. From day 30 onward no doses of jaman fruit, jaman seed and placebo were given. But blood sample were taken to see whether the effect of jaman fruit which was stopped at day 30 (10 days earlier) still sustained or not.

Various doses of Jaman fruits (6no, 12no, 18no) per individuals per day were assigned to groups 1, 2 and 3 for 30 days. From day 30 to day 40 no fruit were given. The dose 6no of jaman fruit were spread over the day as (2 Fruit Morning + 2 Fruit After noon + 2 Fruit in the evening), a dose 12 fruit per day per
individuals (4 Fruit Morning + 4 Fruit After noon + 4 Fruit in the evening), and a dose 18 fruit per day per individuals (6 Fruit Morning + 6 Fruit After noon + 6 Fruit in the evening).

The jaman seed doses 1g, 3g and 6g/day were given to groups 1, 2 and 3 for 30 days respectively. Similarly, the doses of placebo 1g, 3g, and 6g/day were also given to groups 7, 8 and 9 respectively for 30 days. The 1g doses of jaman seed and placebo were spread over the day as 1g (0.350mg breakfast + 350 mg lunch + 300mg dinner) for 30 days respectively. On similar pattern the remaining doses of jaman seed and placebo were given in 3g/day (2g + 2g + 2g) and 6g/day (3g+3g+3g) respectively. All the doses were weigh and then wrapped in a paper.

3.8 DURATION OF THE TRIAL

The total duration for jaman fruit and jaman seed experiment was 30 days and the individuals were monitored until the fortieth day of the experiments. The last 10 days were for the observation on the individuals to study the after effects of this particular fruits and on the diabetic individuals.

3.9 MICRONUTRIENT CONCENTRATION OF JAMAN FRUIT AND SEED

The micronutrient concentration of Jaman fruit and jaman seed were determined by ammonium bicarbonate DTPA or AB_DTPA method. Take 10 g ground sample and diluted with 20 ml AB-DTPA solution and the nutrients were analysed by atomic absorption spectrophotometer. Sample were analysed for
micronutrients (Cu, Zn, Pb, Cr, Fe, Mn, Ni, Cd) by the method as described by (Soltanpor and Schawab 1977).

3.10 COLLECTION OF BLOOD SAMPLE AND BIOCHEMICAL ANALYSIS

Approximately 5 ml fasting blood samples were taken from each diabetic individuals on day 0, 20, 30, and 40. Blood samples were transferred to sterilized centrifuge tubes and allowed to clot at room temperature for 10-15 minutes. After clotting the blood sample were centrifuge for 15-20 minutes at 1500-2500 rpm for serum separation. During serum separation in some sample serum were not clearly separated so the clot need be broken by sharp needle and re-centrifuge that sample for 15 minutes. Serum were separated and stored in freezer at 0C for later analysis of glucose, TGL, Total, high and low-density lipproteins (HDL and LDL) cholesterol. These analysis were carried out in the department of Human Nutrition, Laboratory by using spectrophotometer (S-200D).

3.11 PROCEDURE FOR TESTS

Determination of Glucose

Glucose was determined by the enzymatic colorimetric method of Barham, et al., (1972) and Leoline Kit was used. The enzymatic reaction was occurred in two step: 1. Glucose is oxidized to gluconic acid and hydrogen peroxide in the presence of glucose oxidase enzyme. step: 2. Red quinine is formed in the presence of peroxidase enzyme. The absorbency of the colored substance is taken and the concentration of glucose is calculated. The enzymatic
reactions are as follow.

\[
\text{GOD} \quad \text{Glucose} + \text{O}_2 \quad \text{Glutamic acid} + \text{H}_2\text{O}_2
\]

\[
2\text{H}_2\text{O}_2 + \text{aminoantipyrine} + \text{phenol} \quad \text{Chinoinmine} + 4 \text{H}_2\text{O}
\]

Sample

Serum free of hemolysis

Reagents

**Reagent Solution (R1)**

Concentration

<table>
<thead>
<tr>
<th>Component</th>
<th>Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phosphate buffer (pH 7.5)</td>
<td>250 mmol/l</td>
</tr>
<tr>
<td>Phenol</td>
<td>5 mmol/l</td>
</tr>
<tr>
<td>4-amoinoantipyrine</td>
<td>0.5 mmol/l</td>
</tr>
<tr>
<td>Glucose oxidase (GOD)</td>
<td>&gt; 10 KU/l</td>
</tr>
<tr>
<td>Peroxidase (POD)</td>
<td>&gt; 1 KU/l</td>
</tr>
</tbody>
</table>

**Standard**

<table>
<thead>
<tr>
<th>Component</th>
<th>Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>100 mg/dl</td>
</tr>
<tr>
<td></td>
<td>5.55 mmol/l</td>
</tr>
</tbody>
</table>

**Procedure**

Spectrophotometer (S-200D) was first calibrated for 30 minutes and its wavelength was adjusted at Hg 500 nm, at a temperature 37 °C. 1 cm light path test tubes were used. 10µl of sample and 1000µl of reagent were taken and mixed. Incubate for 20 minutes at 20-25°C or 10 min. at 37°C. Read absorbance against the blank within 60 mins.
Calculation

\[
\text{Glucose concentration} = \frac{\text{standard concentration} \times E_{sa} \text{ (mg/dl)}}{E_{sl} \text{ (mmol/l)}}
\]

Determination Of Triglycerides

Triglycerides were determined by the enzymatic colorimetric method of Gowen et al. (1983) and Linear Chemicals Kit were used.

PRINCIPLE

The triglycerides in the sample are hydrolyzed enzymatically to glycerol and fatty acids. The glycerol formed is converted to glycerol phosphate by Glycerol kinase. Glycerol phosphate is oxidized to dihydroxyacetone phosphate by Glycerol phosphate oxidase. The liberated hydrogen peroxide is detected by a chromogenic acceptor, chlorophenol-ampyrene, in the presence of peroxidase. The red quinone formed is proportional to the amount of triglycerides present in the sample.

\[
\begin{align*}
\text{T} & \text{riglycerides + H}_2\text{O} \xrightarrow{\text{GK}} \text{glycerol + fatty acids} \\
\text{Glycerol + ATP} & \xrightarrow{\text{GPO}} \text{glycerol-3-phosphate} + \text{ATP} \\
\text{Glycerol-3-phosphate + O}_2 & \xrightarrow{\text{HPO}} \text{Dihydroxyacetone phosphate + H}_2\text{O}_2 \\
2\text{H}_2\text{O}_2 + 4\text{-aminophenazone} + 4\text{ dichlorophenol} & \rightarrow \text{quinoneimine} + \text{HCl} + 4\text{H}_2\text{O}
\end{align*}
\]

Sample

Serum free of hemolysis
Reagents

1. Buffer

Pipes buffer 40 mmol/l, pH 7.6
4-chloro-phenol 5.5 mmol/l
Magnesium-ions 17.5 mmol/l

2. Enzyme Reagent

4-aminophenazone 0.5 mmol/l
ATP 1.0 mmol/l
Lipases 150 U/ml
Glycerol-Kinase 0.4 U/ml
Glycerol-3-phosphate oxidase 1.5 U/ml
Peroxidase 0.5 U/ml

3. Standard

2.29 mmol/l (200 mg/dl)

Preparation Of Solution

1. Buffer

Contents of buffer are ready to use and stable up to expiry date when stored at +2 to +8°C.

2. Enzyme reagent

One vial of enzyme reagent 2 was reconstructed with 15 ml of buffer 1 and is stable for 21 days at +2 to +8°C.

3. Standard is ready to use and stable up to expiry date at +2 to +8°C.
Procedure:

The working reagent was prepared by reconstitute one vial of enzymatic reagent (R2) with one bottle of buffer reagent (R1). This working reagent is stable for 4 weeks at 2-8 °C (or 1 week at 15-25 °C). Mix gently to dissolve contents. Colorimeter wavelength was adjusted at Hg 546 nm and temperature was set at 37°C. 1 cm light path cuvettes were used. 20μl sample and 1000μl reagent was taken and mixed. After incubation for 5 minutes the absorbance of the sample (A_{sample}) and standard (A_{standard}) were recorded against the reagent blank within 60 minutes.

Calculation

\[
\text{Triglycerides concentration} = \frac{\bar{A}_{sample}}{\bar{A}_{standard}} \times \text{conc. Of standard} = \text{mg/dL}
\]

Determination of Total Cholesterol

Cholesterol was determined by enzymatic spectrophotometric method of Richmond (1973) and Ecoline Kit was used. In this method the cholesterol is determined after enzymatic hydrolysis and oxidation. The indicator quinoneimine is formed from hydrogen peroxide and 4-aminoantipyrine in the presence of phenol and peroxidase.

\[\text{Cholesterol-ester + } H_2O \xrightarrow{\text{Cholestrol esterase}} \text{Cholesterol + Fatty acids}\]

\[\text{Cholesterol + } O_2 \xrightarrow{\text{Cholestrol oxidase}} \text{Cholestene-3-one + } H_2O\]

\[2H_2O_2 + \text{phenol + 4-Aminoantipyrine} \xrightarrow{\text{Peroxidase}} \text{Quinoneimine + } H_2O\]
Sample

Serum free of hemolysis

Reagent Composition

Reagent

4-Aminoantipyrine 0.30 mmol/l
Phenol 6 mmol/l
Peroxidase ≥ 0.5 U/ml
Cholesterol esterase ≥ 0.15 U/ml
Cholesterol oxidase ≥ 0.1 U/ml
Pipes buffer 80 mmol/l; pH 6.8
Standard 5.17 mmol/l (200 mg/dl)

Preparation of Reagent

The contents are ready to use, and stable up to the expiry date, when stored at ± 2 to ±8°C, in the absence of contamination, protected from light. Standard is ready for use and stable up to expiry date when stored at ±2 to ±8°C.

Procedure

The wavelength was adjusted at 546 nm and the temperature set at 37°C. 1 cm light path cuvettes were used. 10μl sample and 1000μl reagent were pipetted into the cuvette, mixed and incubated for 10 minutes. The absorbance of the sample was measured against blank with in 60 minutes.
Calculation

\[
\text{Conc. Of cholesterol in sample} = \frac{A_{\text{sample}}}{A_{\text{calibration}}} \times \text{conc. Of standard}
\]

**Determination Of HDL Cholesterol**

The CHOD-PAP method was used for the determination of HDL cholesterol. The principal of this method is that the low-density lipoproteins (LDL) and chylomicron fractions are precipitated quantitatively by the addition of phosphotungstic acid in the presence of magnesium ions. After centrifugation, the cholesterol concentration in the HDL (High-density lipoprotein) fraction, which remains in the supernatant, is determined.

**Preparation of Solution**

In semi-micro assays the 80 ml bottle of precipitating reagent was prediluted with 20 ml of redistilled water, stable up to the expiry date specified when store at 15 to 25°C.

**Procedure**

1. **Precipitation**

200 µl sample and 500 µl diluted precipitant were pipetted into centrifuge tube, mixed and allowed to sit for 10 minutes at room temperature, the centrifuged for 10 minutes at 4,000 rpm. The clear supernatant was separated off within 2 hours and the HDL cholesterol was determined by the CHOD-PAP method. In the Cholesterol-PAP method the wavelength was
adjusted at 11g 546 nm and temperature was set at 37°C. 1 cm light path cuvettes were used. 100 µl sample and 1000 µl reagent were pipetted into test tubes and mixed. After 5 minutes of incubation, the absorbance of sample (A_{sample}) and standard (A_{standard}) against reagent blank were recorded with in 60 minutes.

**Calculation**

Concentration of HDL Cholesterol in supernatant = 
\[
\frac{\Delta A_{sample}}{\Delta A_{standard}} \times \text{conc. of standard} = \text{mg/dl}
\]

**Determination Of LDL Cholesterol**

The LDL cholesterol was determined by the following formula:

\[
\text{LDL cholesterol (mg/dl)} = \text{Total Cholesterol} - \frac{\text{Triglycerides}}{5} - \text{HDL Cholesterol} = \text{mg/dl}
\]

**3.12 STATISTICAL ANALYSIS**

The data was statistically analyzed by analysis of variance by using statistical software (Statgraphic Statistical Package, STSC, Statistical Graphics Incorporation, version 2.6, 1987).
4. RESULTS AND DISCUSSION

To see the effects of jaman on blood glucose and lipid profile in diabetic individuals, a clinical trial was conducted. Jaman effects were measured on 108 (54 M and 54 F) Type-II diabetic individuals (Non-Insulin dependent) for 40 days. The ages of the selected individuals were 40-60 years. They were on hypoglycemic drugs rather than insulin therapy (Injection). Their fasting blood glucose and triglyceride were in the range 140-250 mg/dl and 200-250mg/dl respectively. These individuals continued to use their routine food and diabetic medicine i.e. Daonil, Glucophage and Plighiben as prescribed or instructed by their physicians.

The one hundred and eight diabetic individuals were divided into 3 major groups (Jaman Fruit, Jaman Seed and Placebo). Each major group was further divided into 3 sub-groups containing 6 male and 6 female diabetic individuals. Sub-group-1 refers to a dosage of 6 Jaman, Sub-group-2 refers 12 Jaman and sub-group-3 refers 18 Jaman per day per individuals. The Jaman dosages were equally divided in to three diets i.e. breakfast, lunch and dinner. The blood samples were taken at five different stages i.e. day 0 (before the intake of Jaman), 10, 20, 30 and 40 days after the start of the intake of Jaman. The collected data was statistically analyzed by analysis of variance by using statistical software (Stat graphic Statistical Package, STSC, Statistical Graphics Incorporation, version 2.6, 1987). The results of the experiments are presented in forth coming section.
4.1 EFFECT OF JAMAN ON BLOOD GLUCOSE

Glycemic Response of Diabetic Individuals to Jaman Fruit:

The effect of different number of Jaman fruit on the blood glucose levels of diabetic individuals was studied. These individuals were fed with different doses (6, 12, 18) Jaman fruits per day per individuals. The blood samples were collected before intervention day 0 (control), during intervention (days 10, 20 and 30) and after the intervention (day 40) of the experiment. The statistical analysis shows that blood glucose values were non-significantly different. The effect of different doses of Jaman fruits on % reduction of blood glucose of diabetic individuals is given in Appendix-1. The values indicate the mean fasting blood glucose values of groups-1 (6 Jaman per day per individual), group-2 (12 Jaman per day per individual) and group-3 (18 Jaman per day per individual).

Before the intake of Jaman fruit, the mean fasting blood glucose of group-1, was 188.6 mg/dl which reduced 1.2 ±1.1, 2.7 ±3.0, 4.0±4.4, and 1.5 ±1.7% after 10, 20, 30, and 40 days. In group-2 data was 228.5±17.8 mg/dl which reduced 1.0±5.1%, 1.8±6.2, 3.4±6.5, and 1.8 ±5.7% after 10, 20, 30 and 40 days. In group-3 blood glucose was recorded as 223.3 mg/dl on day 0, which reduced 0.5±4.3%, 2.1±4.0, 3.6±5.4 and 1.7±4.4% after 10, 20, 30 and 40 days (Appendix-1).

The effect of different doses of Jaman fruit on blood glucose in male and female is shown in Table-1.
The values without superscripts with the rows are not statistically significant.

| Fasting Blood Glucose (mg/dl) | 177.3±2.1  | 171.1±2.2  | 174.8±2.3  | 192.2±2.9  | 222.6±5.1  | 221.3±7.3  | 223.3±6.7  | 223.8±6.7  | 225.5±8.6  | 221.8±5.7  | 227.2±6.0  | 232.3±7.5  | 235.6±5.4  | 238.8±5.3  | 231.7±7.5  | 232.5±15.0  | 214.2±29.4  | M           |
|------------------------------|------------|------------|------------|------------|------------|------------|------------|------------|------------|------------|------------|------------|------------|------------|------------|------------|------------|------------|------------|
| After Intervention          |            |            |            |            |            |            |            |            |            |            |            |            |            |            |            |            |            |            |            |
| Before Intervention         |            |            |            |            |            |            |            |            |            |            |            |            |            |            |            |            |            |            |            |

**Table 1:** Effect of different doses of Jaman Fruit on blood glucose in diabetic individuals.
The mean fasting blood glucose of diabetic individuals of group-1 was 180 mg/dl and 197.3 mg/dl for male and female respectively (Table-1). In the same individuals reduced to 174.8 mg/dl and 192.2 mg/dl (2.8 to 2.3%). Further intake for 10 days, Jamun fruits reduced the mean fasting blood glucose to 171.1 mg/dl and 191.0 mg/dl (4.7 to 2.7%) recorded on day 30. After the termination of intake of Jamun fruit (recorded on 40th day) the mean fasting blood glucose of both male and female increased to 177.3 mg/dl and 194.2 mg/dl (1.3 to 1.5%) in comparison to the data recorded on day 30.

The data for mean fasting blood glucose of group-2 (12 Jamun fruit per day per individual) ranged from 226.3 mg/dl and 230.6 mg/dl for male and female respectively on day 0 (control). The intake of 4 Jamun fruit per diet of each individuals for 10 days reduced fasting blood glucose to 223.3 mg/dl and 228.6 mg/dl (1.3 to 0.8%) for male and female respectively (Table-1). Similarly, blood glucose education in both male and female 221.3 mg/dl and 227.1 mg/dl (2.2 to 1.5%) and 218.0 mg/dl and 223.3 mg/dl (3.6 to 2.7%) were recorded on 20th day and 30th day respectively. After 10 days of the termination of Jamun intake, the mean fasting blood glucose recorded on 40th day increased in both male and female (222.6 mg/dl and 225.8 mg/dl (with an increase of 1.3 to 1.6%), respectively.
The mean fasting blood glucose of group-3, diabetic individuals (18 Jaman fruit per day per individuals; Table-1) for male and female ranged from 214.2 and 232.5 mg/dl, respectively recorded before the intake of Jaman fruits. The mean fasting blood glucose levels reduced 212.5 and 231.7 mg/dl (0.9 to 0.3%) on day 10, 208.8 and 228.5 mg/dl (2.5 to 1.7%) on day 20 and 205.3 and 225.0 mg/dl (4.1 to 2.2%) on day 30, for male and female respectively. On day 40 (10 day after the termination of Jaman dosage) the levels of blood glucose increased 211.8 and 227.2 mg/dl (1.1% and 2.2%) for male and female respectively.

Our results showed that the intake of Jaman fruit (6, 12 & 18 jaman per day per individuals for both male and female) non-significantly reduced the mean fasting blood glucose. The decreasing trend continued from the first reading to the last reading during Jaman intake. In our experiment Jaman fruit could have increased glucose utilization that could be the reason for lowering of blood glucose in diabetic individuals. Our results are in conformity to the earlier reported hypoglycemic response of Jaman pulp extract on animals (Achrekar et al., 1991). Similarly, Arbab et al., (1989) tested Jaman leaves on normal and hyper/glycaemic adult rabbits and human. They found that 20 and 40 mg/kg Jaman ethanol extract significantly reduced the blood sugar.

Ahmed et al., (1995) confirmed the hypoglycemic action of jaman extract in animal and reported that it reduced glucose level (2.8 mg/ml to 3.0 mg/dl). The utilization of herbal medicine for healthcare is in use for a long time (Farnsworth et al., 1977). Anita et al., (1990) reported that raw bittergourd, Jambu and fenugreek seed significantly reduced fasting blood
glucose levels in diabetic patients. Moreover, Goodcare (Pharma PVT. LTD., 1990) found that different herbs and Jaman granules had positive response in lowering blood glucose. Furthermore, these granules delayed the rate of absorption of carbohydrates in gastro-intestinal transit and increased the uptake of glucose by cells. Further, Harry (1992) reported that Jaman contained Flavonoids and phenolic compound, which are effective in treatment of diabetes. Bratman (2000) reported that American and Asian ginsing both lower blood sugar in Type-II diabetic individuals. The ginsing is safe and effective. Further, he pointed out that these herbs enhance the release of insulin from the pancreas and increase the number of insulin receptors.

However, the herbal medicines have potency to increase the production of insulin, and rejuvenate the liver and pancreas.

The blood glucose levels increased after the discontinuation of Jaman intake confirming a relationship or interaction of Jaman fruit with insulin production. Since, Jaman fruit contains arginine and many other essential amino acids (Noomr et al., 1996), which have a positive role in the regulation of insulin secretion (Campe et al., 1994).

4.2 GLYCEMIC RESPONSE OF DIABETIC INDIVIDUALS TO JAMAN SEED

The effect of different doses of Jaman seed on blood glucose in different groups of diabetic individuals are presented in Appendix-II.
The values indicate the mean fasting blood glucose values of group-1 (1 gm Jaman seed per day per individual), group-2 (3 gm Jaman seed per day per individual) and group-3 (6 gm Jaman seed per day per individual).

Before the intake of Jaman seed, the mean fasting blood glucose of group-1, was 227.8 mg/dl which increased 0.2 ±1.2%, on day 10 and then slightly reduces on 1.0 ±2.1, 1.7±0.2% on day 20, 30 but the blood glucose of the same individuals again increased (2.0%) on day 40. In group-2, diabetic individuals the mean fasting blood glucose was 168.7 mg/dl which reduced 5.1±2.1%, 3.0±0.4, 3.0±1.1, 3.0±2.0% after 10, 20, 30, and 40 days. In group-3, individuals blood glucose was recorded as 157.10 mg/dl which consistently reduced 2.7±0.4%, 2.1±1.1, 2.1±0.5 and 2.6±2.1% after 10, 20, 30 and 40 days (appendix-II).

The effect of different doses of Jaman seed on blood glucose in male and female diabetic individuals are presented in Table-2.
The values without superscripts with the rows are not statistically significant.

3* = 6 g Jamun seed fed to the diabetic individuals per day.

2* = 3 g Jamun seed fed to the diabetic individuals per day.

1* = 1 g Jamun seed fed to the diabetic individuals per day.

<table>
<thead>
<tr>
<th>Dose of Jamun (g/day)</th>
<th>Mean ± SD</th>
<th>Median ± SD</th>
<th>Day 0</th>
<th>Day 20</th>
<th>Day 30</th>
<th>Day 40</th>
<th>Day 50</th>
<th>Day 60</th>
<th>Day 70</th>
<th>Day 80</th>
<th>Day 90</th>
<th>Day 100</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.2</td>
<td>151.7±15.4</td>
<td>151.7±15.4</td>
<td></td>
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Table 2: Effect of different doses of Jamun seed on fasting glucose in diabetic individuals.
Before the intake of jaman seed, the mean fasting blood glucose of group-I was 165.7 and 149.7 mg/dl for male and female respectively (Table-2). After the intake of jaman seed (1 gram per day) for 10 days, the mean fasting blood glucose level of male individuals was increased 166.7 mg/dl (0.6%) but in female diabetic individuals blood glucose was reduced 149.3 mg/dl (0.2%) respectively. With further intake for 10 days the blood glucose of the same individuals reduced 165.8 and 146.6 mg/dl (0.12 - 2.1 %). Further intake for 10 days jaman seed reduced the mean fasting blood glucose to 164.5 and 145.2 mg/dl (0.9-2.5%) recorded on day 30. after the termination of dose, of jaman seed, data recorded on 48th day, the mean fasting blood glucose of male and female increased 172.2 and 149.5 mg/dl (4.32-0.3%), in comparison to the data recorded during the intake of jaman dosages.

Whereas this data was also increased in comparison to the intervention period and higher than the control day 0 (6.5% for male individuals while it was slightly reduced (0.2%) for female diabetic individuals.

Regarding the data of mean fasting blood glucose of group-2, diabetic individuals (3 gm Jaman seed per day per individuals) ranged from 171.0 and 166.8 mg/dl for male and female respectively (Table. 2). The intake of 3 gm Jaman seed per day of each individuals for 10 days reduced fasting blood glucose 164.0 and 156.8 mg/dl (4.1 to 6.1%) for male and female respectively (Table-2). The blood glucose was again increased in both male and female 167.1 and 160.7 mg/dl (2.4 to 3.8%). The values on day 30 of the feeding trial were similar to the values on
day 20 in male diabetic individuals. The fasting blood glucose values on day 30 were also increased 164.0 mg/dl (1.8%) respectively.

After 10 days of the termination of Jaman seed dose intake, the mean fasting blood glucose recorded on 40th day increased in both male and female 162.2 and 165.5 mg/dl with an increase (3.5 to 0.8%), respectively.

Before the intake of Jaman seed, the mean fasting blood glucose of group-3, was 154.0 and 160.2 mg/dl for male and female respectively (Table-2). After the intake of Jaman seed (6gm per day) for 10 days, the mean fasting blood glucose levels was reduced 147.0 and 158.6 mg/dl (0.8 to 1.3%) in male and female respectively. With further intake for 10 days, the blood glucose of the same individuals in male was increased 152.4 mg/dl (0.9%). But the mean fasting value 155.0 mg/dl (1.0%) on day 20 was lower. The value on day 30 in male individuals again reduced 146.6 mg/dl (1.2%) but the values on same day in case of female diabetic individuals were further increased 159.2 mg/dl (1.0%).

After the termination of jaman seed intake (recorded on 40th day) the mean fasting blood glucose of both male and female increased 182.6 and 194.4 mg/dl (018.5 to 21.3%) in comparison to the data recorded during the intake of Jaman seed dosages.

The results of Jaman seed experiments 1, 3 and 6 g per day per individuals for male and female showed reduced mean fasting blood glucose non-significantly in diabetic individuals. The decreasing trend in hypoglycemic response continued with the increased in duration of jaman seed intake. In
Jaman fruit and jaman seed experiment both may have increased glucose utilization that could be the reason for lowering of blood glucose in diabetic individuals. The finding of this experiment are in line with the previous results of hypoglycemic response of Jaman seed (Ashok et al, 2001). Similarly, Virmani et al (2001) reported that Jaman seed powder decreased the fasting blood glucose 6.99%. Further they reported that jaman dosages after meal reduced the blood sugar levels (10.56%). Arbab et al., (1989) also tested Jaman leaves on human and found that 20 and 40 mg/kg Jaman ethanol extract significantly reduced the blood sugar. However, Dewan (2000) advised a dose of 2-3 gm a day jaman seed powdered for the diabetic patients. The jaman seed contained a glycoside, which prevents the conversion of starch into sugar.

The hypoglycemic action of jaman extract in animal reduced glucose level from 161 ± 2.8 mg/ml to 134 ± 3.0 mg/dl (Ahmed et al, 1995). Chemicals like Flavonoids and phenolic compounds are contained in jaman seed that help in treatment of diabetes (Harry, 1992). Goodcare Pharma pvt. Ltd (1990) prepared granules from different herbs e.g., gujar, neem, jamun, karella, giloi, khair, haldi, amla, vijaysar, Vijaysar, Tejpatta, Gular, Kutki, Methi, Purified Shilajeet, Powderang Bhasma, Yasad Bhasma in various quantities. They observed a positive response of these granules in reduction of blood glucose level of diabetic individuals. In addition, these granules also delayed the rate of absorption of carbohydrates in gastro-intestinal transit and increased the uptake of glucose by cells.
Dietrich (1974) tested aqueous extracts of fresh Jaman seeds on diabetic rabbits and found that significantly reduced fasting blood sugar (15 to 35%) in four to five hours. He also reported that Jaman contained triterpene acids, oleanolic acid and crategelic acid that may also help in control of diabetes.

The blood glucose levels increased after the discontinuation of Jaman seed intake confirming a relationship or interaction of Jaman seed with insulin production. However, the herbal medicine contains various chemicals that may help in prevention of diabetes.

4.3 EFFECT OF PLACEBO ON BLOOD GLUCOSE IN DIABETIC INDIVIDUALS

As mentioned earlier, these experiment were conducted on the diabetic individuals and were self control. However, placebo trial were conducted in similar pattern in order to see the psychological effect of the jaman seed. The content of the placebo was just maize flour in the similar doses as provided the jaman seed i.e. 1, 3, and 6 gm per day per individuals.
The values without superscript are not statistically significant.

*3 = 6 g Placebo (Maltex Flour) led to the diabetic individuals per day.

*2 = 3 g Placebo (Maltex Flour) led to the diabetic individuals per day.

*1 = 1 g Placebo (Maltex Flour) led to the diabetic individuals per day.

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<tr>
<td>SD</td>
<td>53.7±1.6</td>
<td>3.9±1.8</td>
<td>52.3±2.0</td>
<td>123±7.5</td>
<td>143±7.2</td>
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</tbody>
</table>

|       | 198±0.5    | 20.5±0.2   | 196±0.3  | 216±2.0   | 214±2.3 | 0   |
| Mean  | 137±4.4    | 112±2.1    | 97±4.5   | 208±7.3    | 206±7.3 | 1   |
| SD    | 53.7±1.6   | 3.9±1.8    | 52.3±2.0 | 123±7.5    | 143±7.2 | 1   |

Table 3: Effect of Placebo on Blood Glucose Level in Diabetic Individuals

<table>
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<tr>
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<tr>
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5. EFFECT OF JAMAN ON TRIGLYCERIDE OF DIABETIC INDIVIDUALS

5.1 HYPOLIPIDEMIC RESPONSE OF DIABETIC INDIVIDUALS TO JAMAN FRUIT

The effects of different doses of Jaman fruit on fasting blood triglyceride (TGL) levels of diabetic individuals were non-significantly different.

The mean values of the fasting blood TGL of groups-1 (6 Jaman per day per individual), group-2 (12 Jaman per day per individual) and group-3 (18 Jaman per day per individual) are presented in Table-4.

Before the intake of Jaman fruit, the mean fasting blood TGL of group-1, diabetic individuals was 225.9 mg/dl which reduced 0.8±2.0%, 1.9±4.1, 2.8±1.2% and increased 0.2±1.9% after 10, 20, 30, and 40 days. In group-2, individuals data was 227.5±9.2 mg/dl which reduced 0.6±1.1%, 0.3±3.0, 2.1 ± 4.4% and increased 0.13% after 10, 20, 30 and 40 days. Similarly in group-3, individuals TGL was noted as 230.3 mg/dl which reduced 3.5±11.3%, 5.3 ±11.3, 6.6±10.4 and 4.6±12.6 % after 10, 20,30, and 40 days (Appendix-III).
The values without superscripts will the rows are not statistically significant.

3* = 18 Jamun fruit fed to the diabetic individuals per day.
2* = 12 Jamun fruit fed to the diabetic individuals per day.
1* = 6 Jamun fruit fed to the diabetic individuals per day.

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</table>

Fasting Blood TRC (mg/dl)

Table 4 Effect of different doses of Jamun fruit on blood TRC in diabetic individuals.
The mean fasting blood TGL of group-1, diabetic individuals was 227.1 and 224.4 mg/dl for male and female respectively (Table-4). After the intake of Jaman fruit (6 jaman per day) for 10 days, the mean fasting blood TGL levels of these individuals reduced to 225.3 and 222.8 mg/dl (0.7 to 0.8%) for male and female respectively. With further intake of jaman fruit for 10 days, the blood TGL of the same individuals reduced to 223.1 and 220.1 mg/dl (1.7%). Further intake for 10 days, Jaman fruits reduced the mean fasting blood TGL to 219.5 mg/dl (3.3 to 2.0%) recorded on day 30. After 10 day of the termination of Jaman fruit intake (recorded on 40th day) the mean fasting blood TGL of both male and female increased to 227.5 and 225.5 mg/dl (0.2 to 0.3%) in comparison to the data recorded on day 30.

The TGL values of the diabetic individuals of group-2 was 183.6 and 192.3 mg/dl for male and female respectively (Table 4). After the intake of Jaman fruit (12 jaman per day per individual) for 10 days, the mean fasting blood TGL levels reduced to 180.2 and 190.4 mg/dl (1.8 to 0.9%) for male and female respectively. With further intake for 10 days, the blood TGL of the same individuals reduced 177.8 and 187.5 mg/dl (3.1 to 2.1%). Further intake for 10 days, Jaman fruit reduced the mean fasting blood TGL to 172.6 and 187.1 mg/dl (5.9 to 2.1%) recorded on day 30. After 10 days of the termination of jaman intake (on day 40) the mean fasting blood TGL of both male and female increased to 180.6 and 189.4 mg/dl (1.5 to 1.3%) (Table-4) in comparison to the data recorded on day 30.
The fasting blood TGL of diabetic individuals of group-3 was 232.0 and 228.7 mg/dl for male and female respectively (Table-4). After the intake of Jaman fruit (18 jaman per day per individual) for 10 days, the mean fasting blood TGL levels reduced to 212.5 and 231.7 mg/dl (8.4 to 1.3%) for male and female respectively. With further intake for 10 days, the blood TGL of the same individuals reduced to 208.8 and 228.5 mg/dl (10.1 to 0.8%). Further intake for 10 days, Jaman fruit reduced the mean fasting blood TGL to 205.2 and 225.0 mg/dl (11.5 to 1.6%) recorded on day 30. After 10 days of the termination of Jaman intake (recorded on 40th day) the mean fasting blood TGL of both male and female increased to 211.8 and 227.2 mg/dl (9.0 to 1.6%) in comparison to the data recorded on day 30.

The results of Jaman seed experiments (1, 3 & 6 g) per day per individuals for male and female showed reduced mean fasting blood glucose non-significantly in diabetic individuals. The decreasing trend in hypoglycemic response continued with the increased in duration of jaman seed intake. In jaman fruit and jaman seed experiment both may have increased glucose utilization that could be the reason for lowering of blood glucose in diabetic individuals. The finding of this experiment are in line with the previous results of hypoglycemic response of Jaman seed (Ashok et al, 2001). Similarly, Virmani et al (2001) reported that Jaman seed powder decreased the fasting blood glucose 6.99%. Further they reported that jaman dosages after meal reduced the blood sugar levels (10.56%). Arbab et al., (1989) also tested Jaman leaves on human and found that 20 and 40 mg/kg Jaman ethanol extract
significantly reduced the blood sugar. However, Dewan (2000) advised a dose of 2-3 gm a day jaman seed powdered for the diabetic patients. The jaman seed contained a glycoside, which prevents the conversion of starch into sugar.

The hypoglycemic action of jaman extract in animal reduced glucose level from $161 \pm 2.8 \text{ mg/ml}$ to $134 \pm 3.0 \text{ mg/dl}$ (Ahmed et al., 1995). Chemicals like Flavonoids and phenolic compounds are contained in jaman seed that help in treatment of diabetes (Harry, 1992). Goodcare Pharma Pvt. Ltd (1990) prepared granules from different herbs e.g., gummar, neem, jamun, karella, giloi, khair, haldy, amla, vijaysar, Vijaysar, Tejpatta, Gular, Kutki, Methi, Purified Shilajeet, Powderaug Bhasma, Yasad Bhasma in various quantities. They observed a positive response of these granules in reduction of blood glucose level of diabetic individuals. In addition, these granules also delayed the rate of absorption of carbohydrates in gastro-intestinal transit and increased the uptake of glucose by cells.

Dietrich (1974) tested aqueous extracts of fresh Jaman seeds on diabetic rabbits and found that significantly reduced fasting blood sugar (15 to 35%) in four to five hours. He also reported that Jaman contained triterpene acids, oleanolic acid and crategelic acid that may also help in control of diabetes.

The blood glucose levels increased after the discontinuation of Jaman seed intake confirming a relationship or interaction of Jaman seed with insulin production. However, the herbal medicine contains various chemicals that may help in prevention of diabetes.
5.2 HYPOLIPEDEMIC RESPONSE OF DIABETIC INDIVIDUALS TO JAMAN SEED

The effects of different doses of Jaman seed on fasting blood triglyceride (TGL) levels of diabetic individuals were non-significantly different.

The mean values of the fasting blood TGL of groups-1 (1gm Jaman seed per day per individual), group-2 (3gm Jaman per day per individual) and group-3 (6gm Jaman per day per individual) are presented in Appendix-IV.

Before the intake of jaman seed the mean fasting blood TGL of the diabetic individuals of group-1 was 227.8 mg/dl, which dropped 0.4±0.01% on day 10, and increased 0.9±0.2, 0.8±0.2% on day 20 and 30 while the values again increased 0.2±0.01% on day 40. In group-2, individuals the fasting TGL level was 229.9 mg/dl which increased 1.1±1.0%, 1.1±0.3 0.6±0.3 and 0.4±0.1% after 10, 20, 30 and 40 day. The TGL values of group-3, diabetic individuals was 236.3 mg/dl which increased 12.4±7.9%, 2.2±1.2, after 10 and 20 days but TGL values reduced 8.9±3.2 and 8.4±3.4% on day 30 and 40.(Appendix-IV).
The values without superscripts with the rows are not statistically significant.

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<td>229.2 ± 17.2</td>
<td>226.8 ± 17.2</td>
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</table>

Table 5: Effect of different doses of Jamun seed on blood TRC in diabetic individuals.
The mean fasting blood TGL of group-1, diabetic individuals was 214.0 and 241.7 mg/dl for male and female respectively (Table-5). After the intake of Jaman seed (1 gm jaman seed per day per individuals) for 10 days, the mean fasting blood TGL levels of these individuals increased 216.0 (0.9%) in male but reduced 237.7 mg/dl (9.5%) in female respectively. Further intake for 10 days, Jaman seed reduced the mean fasting blood TGL, 215.8 mg/dl (0.8%) but in female TGL values increased 244.3 mg/dl (1.0%). The mean fasting values on day 20 of male diabetic individuals increased 216.5 mg/dl (1.1%) but the female values remain lower 243.0 mg/dl (0.5%) recorded on day 30. After 10 day of the termination of Jaman seed intake (recorded on 40th day) the mean fasting blood TGL of both male and female increased to 213.3 and 241.3 mg/dl (3.2 to 0.1%) in comparison to the data recorded on day 30.

The fasting blood TGL of diabetic individuals of group-2 was 238.8 and 221.0 mg/dl for male and female respectively (Table-5). After the intake of Jaman seed (0.8gm jaman seed per day per individuals) for 10 days, the mean fasting blood TGL levels of these individuals reduced 236.8 mg/dl (4.1%) in male but in female increased 228.5 mg/dl (3.3%) respectively. Further intake for 10 days, Jaman seed increased the mean fasting blood TGL 238.2 mg/dl but in female TGL values increased 286.2 mg/dl (3.8%). The mean fasting values on day 20 of female diabetic individuals reduced 226.8 mg/dl (2.6%). With further use of jaman seed for 10 days the mean fasting TGL values on day 30 reduced 233.7 mg/dl (2.1%) but the mean values of TGL in female 229.2 mg/dl (3.2%) increased. After 10 day of the termination of Jaman seed intake (recorded on 40th day) the
mean fasting blood TGL of male 234.7 mg/dl (2.7%) increased as compared to the values on days 30. The mean fasting values of TGL on day 40 was 227.5 mg/dl (2.9%) lower than the values on day 30 but higher on the value on day 0 (control).

The fasting blood TGL of diabetic individuals of group-3 was 238.0 and 234.2 mg/dl for male and female respectively. After the intake of Jaman seed (6gm per day per individuals) for 10 days, the mean fasting blood TGL levels of these individuals increased 249.6 (+0.8%) in male but in female increased 286.0 mg/dl (22.1%) respectively. Further intake for 10 days, Jaman seed reduced the mean fasting blood TGL 249.6 mg/dl but in female TGL values reduced 232.0 mg/dl (0.9%). The mean fasting values on day 30 of male diabetic individuals reduced 214.4 mg/dl (9.9%) but in the female TGL values reduced 216.3 (7.6.0%) recorded on day 30. After 10 day of the termination of Jaman seed intake the value (noted on 40th day) the mean fasting blood TGL of both male and female were observed unchanged 214.0 and 219.5 mg/dl (8.6% to 6.2%) in comparison to the data recorded on day 30.

Khan et al (1998) reported that *spices-Murraya Koenigii* and *Brassica juncea* reduced the concentrations of cholesterol, TGL and phospholipids. A significant decrease in blood lipids levels have been reported by Bordia et al (1979) when they provided a dose of 2.5 g Fenugreek twice daily for 3 months to diabetic individuals. In another study, Arum et al (1981) reported that garlic significantly lowered serum triglycerides but increase the high-density lipoproteins. Whereas, Mani et al (2000) provided a dose of 2 g/day spirulina
tablet and reported the same significant reduction in blood triglycerides. Our results are similar with those of Baskaran et al (1990). They reported that gurmar extract stimulate insulin secretion, lower triglycerides without side effects. They also observed that the extract was superior than medication for blood glucose stabilization and lowering of triglycerides. Further, they suggest that the supplementation may regenerated/repaird beta cell in diabetic individuals. A study conducted by Gupta et al (1994) on psyllium husk showed significant decrease in triglyceride in diabetic individuals having hyperlipemia. Soni (1992) administered 500 mg of curcumin per day for 7 days to the healthy volunteers and reported that reduced serum triglyceride (33%).

5.3 EFFECT OF PLACEBO ON TRIGLYCERIDE IN DIABETIC INDIVIDUALS

As mentioned earlier, these experiment were conducted on the diabetic individuals and were self control. However, placebo trial were conducted in similar pattern in order to see the psychological effect of the jaman seed. The content of the placebo was just maize flour in the similar doses as provided the jaman seed i.e. 1, 3, and 6 gm per day per individuals.
The values without superscripts with the rows are not statistically significant.

$* = \text{6 g Placebo (Maze Flour) fed to the diabetic individuals per day.}$

$\text{2 g Placebo (Maze Flour) fed to the diabetic individuals per day.}$

$\text{1 g Placebo (Maze Flour) fed to the diabetic individuals per day.}$

<table>
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<td>Day 30</td>
<td>Day 40</td>
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Table 6: Effect of Placebo on Fasting Blood TGL (mg/dl) in Diabetic Individuals.
EFFECT OF JAMAN ON CHOLESTEROL OF DIABETIC INDIVIDUALS

6.1 HYPOCHOLESTEROLEMIC RESPONSE OF DIABETIC INDIVIDUALS TO JAMAN FRUITS

The effects of different doses of Jaman fruit on fasting blood cholesterol levels of diabetic individuals were significantly different.

The mean values of the fasting blood cholesterol of groups-1 (6 Jaman per day per individual), group-2 (12 Jaman per day per individual) and group-3 (18 Jaman per day per individual) are presented in Table-7.

Before the intake of jaman fruit, the mean fasting blood cholesterol of diabetic individuals of group-1, was 225.6 mg/dl which reduced 4.2±4.1%, 7.3±4.8, 10.2±4.9 and 5.8±2.8 after 10, 20, 30, and 40 days. In group-2 data was 207.4 mg/dl which reduced 2.2±5.8%, 4.5±5.7, 6.8±6.3 and 4.9±4.2±9.1% after 10, 20, 30 and 40 days. In group-3 diabetic individuals the fasting blood cholesterol was observed 208.2 mg/dl, which reduced 3.4±3.2%, 6.5 ±4.7, 9.6±5.1 and 6.0±5.0 % after 10, 20,30, and 40 days (Appendix-V).

The mean fasting blood cholesterol values of the selected individuals of group-1 on day 0 were 231.3 and 220.0 mg/dl for male and female respectively (Table-7). After the intake of jaman fruit (6 jaman per day for 10 days, the mean fasting blood cholesterol levels reduced to 218.8 and 213.5 mg/dl (1.7 to 0.9) for male and female respectively. With further intake of jaman fruit for 10 days, the blood cholesterol of the same individuals reduced to 212.7 and 205.5 mg/dl (2.3 to 1.7%). Further intake for 10 days, Jaman fruits reduced the mean fasting blood cholesterol to 206.2 to 199.0 mg/dl (4.7 to 2.7%) recorded on day 30. After 10 day of the termination of jaman fruit intake (recorded on 40th day) the mean fasting blood cholesterol of both male and female increased to 219.2 and 206.0 mg/dl (1.3 to 1.5%) in comparison to the data recorded during the jaman intake.
The values without superscripts with the rows are not statistically significant.

18Jamun fruit feed to the diabetic individuals per day.
12Jamun fruit feed to the diabetic individuals per day.
6 Jamun fruit feed to the diabetic individuals per day. $*=p<0.05$

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**Table 2:** Effect of different doses of Jamun fruit on blood cholesterol in diabetic individuals.
The cholesterol values of the diabetic individuals of group-2 was 201.7 and 213.1 mg/dl for male and female respectively (Table-7). After the intake of jaman fruit (12 jaman per day per individual) for 10 days, the mean fasting blood cholesterol levels reduced to 198.5 and 207.0 mg/dl (0.5 to 2.8%) for male and female respectively. With further intake for 10 days, the blood cholesterol of the same individuals reduced to 195.2 and 201.0 mg/dl (3.2 to 5.6%). Further intake for 10 days, Jaman fruit reduced the mean fasting blood cholesterol to 191.3 and 195.0 mg/dl (5.1 to 8.4%) recorded on day 30. After 10 days of the termination of jaman intake (on day 40) the mean fasting blood cholesterol of both male and female increased to 205.7 and 188.8 mg/dl (1.3 to 11.6%) (Appendix-1.2c) in comparison to the data recorded on day 30.

The mean fasting blood cholesterol of diabetic individuals of group-3 was 195.2 and 221.2 mg/dl for male and female respectively (Table-7). After the intake of Jaman fruit (18 jaman per day per individual) for 10 days, the mean fasting blood cholesterol levels reduced to 192.0 and 210.2 mg/dl (1.6 to 4.9%) for male and female respectively. With further intake for 10 days, the blood cholesterol of the same individuals reduced to 189.5 and 199.7 mg/dl (2.9 to 9.2%). Further intake for 10 days, Jaman fruit reduced the mean fasting blood cholesterol to 184.7 and 191.7 mg/dl (5.3 to 13.3%) recorded on day 30. After 10 days of the termination of Jaman intake (recorded on 40th day) the mean fasting blood cholesterol of both male and female increased to 190.5 and 200.7 mg/dl (1.3 to 2.0 %) in comparison to the data recorded on day 30.
In our results, the response of jamun fruit and jamun seed on reduction of cholesterol was comparatively higher than the other parameters. Anita et al. (2002) reported that jamun, karela, and methi seeds significantly improved serum lipid profile lower total, LDL cholesterol, VLDL cholesterol and triglycerides and increased HDL cholesterol level. Diabetguard granules were made from different herbs e.g., jamun, gurmar, neem, karela, giloi, khair, haldy, amla, vijaysar, Vijaysar, Tejpatta, Gular, Kutki, Methi, Purified Shilajeet, Powderang Bhasma, Yasad Bhasma, showed a positive response in reduction of serum cholesterol and triglyceride level of diabetic individuals. In addition, these granules also delayed the rate of absorption of carbohydrates in gastro-intestinal transit and increased the uptake of glucose by cells (Goodcare Pharma Pvt. Ltd, 1990). Edelberg (1990) reported that Fenugreek seeds reduced cholesterol levels. It is rich in fiber therefore they slow the rate of gastric emptying, inhibit the absorption of glucose in the intestine and lower the cholesterol levels in blood serum. Similarly, Bordia et al. (1979) found that Fenugreek significantly decreased the blood lipids and cholesterol levels. Mahpara and Khan (2002) reported that cinnamon reduced the cholesterol levels in diabetic individuals. They didn't found any reason for the reduction but they have suggested that some constituents of cinnamon may have blocked the synthesis of cholesterol or facilitated the clearance of cholesterol from the body. Purohit et al. (1999) reported that the use of curcuma longa extract in rabbits reduced the serum cholesterol. Khan et al. (1998) fed Murraya Koenigii and Brassica juncea to rats for 3 months. They found that these spices reduced the concentrations of cholesterol, TGL and phospholipids in serum, aorta, liver and
heart. Similarly, Chithra and Leelamma (1997) reported that coriander seeds 
(Coriandrum sativum) decreased the cholesterol in the tissue of rats. Arun et al 
(1981) studied the effect of garlic on diabetic individuals. They reported that 
garlic significantly lowered serum cholesterol and triglycerides.

6.2 HYPOCHOLESTEROLEMIC RESPONSE OF DIABETIC 
INDIVIDUALS TO JAMAN SEED

The effects of different doses of Jaman seed on fasting blood cholesterol 
levels of diabetic individuals were significantly different.

The mean values of the fasting blood cholesterol of groups-4 (1gm Jaman 
seed per day per individual), group-5 (3gm Jaman per day per individual) and 
group-6 (6gm Jaman seed per day per individual) are presented in Table-8.

Before the intake of jaman seed, the mean fasting blood cholesterol of 
group-1, diabetic individuals was 205.3±7.9 mg/dl which reduced 3.9±4.1%, 
4.6±4.8, 4.5±4.9 and 3.4±2.8 after 10, 20, 30, and 40 days. In group-2 the mean 
fasting values of cholesterol was 197.3±35.0 mg/dl which reduced 1.5±1.1, 
0.6±5.7% on 10 and 20 days but an increased of 0.5±8.8% was recorded on 30 
days again 1.3±3.4% reduced on day 40 of the experiment. In group-3, the mean 
fasting cholesterol of the diabetic individuals was 213.0±26.3 mg/dl which 
reduced 11.6±14.8%, 12.2±17.2, 10.7±19.7% after 10, 20, 30 days and an 
increased of 4.7±23.3 % after 40 days was recorded (Appendix-VI).
The values within superscripts with the same rows are not statistically significant.

3* = 6g jamam seed fed to the diabetic individuals per day.
2* = 3g jamam seed fed to the diabetic individuals per day.
1* = 1g jamam seed fed to the diabetic individuals per day.

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Table 8: Effect of different doses of jamam seed on blood cholesterol of diabetic individuals.
The mean fasting cholesterol values of the diabetic individuals of group-1 on day 0 were 187.7 and 224.72 mg/dl for male and female respectively (Table-8). After the intake of jaman seed (1 gm jaman seed per day) for 10 days, the mean fasting blood cholesterol levels reduced to 171.0 and 223.8 mg/dl (8.8 to 0.4%) for male and female respectively. With further intake of jaman seed for 10 days, the blood cholesterol of the same individuals reduced to 170.3 and 219.8 mg/dl (9.7 to 2.6%). Further intake for 10 days, jaman seed increased the mean fasting blood cholesterol 171.2 mg/dl (8.7%) in male and the mean values on 30 in female remain unchanged (7.6%). After termination of the jaman doses for 10 days the mean values of cholesterol was reduced 170.3 mg/dl (9.2%) for male diabetic individuals as compared to the mean values on day 30.

Regarding the mean fasting blood cholesterol of the diabetic individuals of group-2 on day 0 was 172.0 and 223.5 mg/dl for male and female respectively (Table-8). After the intake of jaman seed (3 gm jaman seed per day per individuals) for 10 days, the mean fasting blood cholesterol levels reduced 169.5 and 219.8 mg/dl (1.4 to 1.6%) for male and female respectively. With further intake of jaman seed for 10 days, the blood cholesterol of the same individuals increased 171.3 and 223.3 mg/dl (0.4 to 0.08%). Further intake of jaman seed by the same individuals for 10 days increased the mean fasting blood cholesterol 175.3 mg/dl (1.2%) in male diabetic individuals while in female the mean fasting cholesterol was reduced 222.3 mg/dl (0.5%) on day 30 of the trial.
After discontinuation of the dose for 10 days the mean cholesterol levels was 175.7 mg/dl (2.1%) for male which is similar to the values on day 30 and higher than the values on day 0 (control 0). Similarly the mean values on day 40 for female reduced 216.7 mg/dl (3.0%) as compared to day 30 and day 0 (control 0)

Data of the mean fasting blood cholesterol of the diabetic individuals of group-3 on day 0 was 209.5 and 205.8 mg/dl for male and female respectively (Table-8). After the intake of jaman seed (6 gms jaman seed per day per individuals) for 10 days, the mean fasting blood cholesterol levels reduced 196.8 and 190.0 mg/dl (6.0 to 7.6%) for male and female respectively. With further intake of jaman seed for 10 days, the blood cholesterol of the same individuals reduced 184.0 and 192.0 mg/dl (12.0 to 6.7%). Further intake of jaman seed by the same individuals for 10 days reduced the mean fasting cholesterol 181.3 mg/dl (13.4%), for male on day 30 but the mean fasting cholesterol increased 215.3 mg/dl (4.6%) in female individuals.

After discontinuation of the dose for 10 days the mean cholesterol levels increased 197.2 mg/dl (5.8%) in male diabetic individuals but in female the cholesterol values is also increased 225.5 mg/dl (9.5%) respectively.

Anita et al (2002) reported that karla, jaman, and fenugreek seed significantly lower total, LDL cholesterol, VLDL cholesterol in diabetic individuals. Edelberg (1990) reported that Fenugreek seed’s also lower the cholesterol levels in type-2 diabetic individuals. It is rich in fiber and slow the rate
of gastric emptying, inhibits the absorption of glucose in the intestine and lowers the cholesterol levels. Our results are in conformity with the earlier reported hypocholesterolemic response of Jaman seed (Goodcare Pharma Pvt. Ltd, 1990). In another study, Purolat et al (1999) tested curcuma longa extract (50% EtOH) in hyperlipidaemic rabbit and found that reduced significantly serum cholesterol levels. Khan et al (1998) reported that 2 common spices- Murraya Koenigii and Brassica juncea reduced the concentrations of cholesterol, TGL and phospholipids in serum. These spices play a significant role in controlling the development of hypercholesterolaemia and arteriosclerosis. Similarly, Chithra and Leelamma (1997) reported that coriander seeds (Coriandrum sativum) also reduced the level of cholesterol. Maphara and Khan (2002) reported that cinnamon has lasting hypocholesterolemic effect in diabetic individuals. Spices and natural products have an effect on cholesterol in humans. Curry leaf and mustard seeds decreased total serum cholesterol in rats (Khan et al, 1996).

6.3 **EFFECT OF PLACEBO ON CHOLESTEROL IN DIABETIC INDIVIDUALS**

As mentioned earlier, these experiment were conducted on the diabetic individuals and were self control. However, placebo trial were conducted in similar pattern in order to see the psychological effect of the jaman seed. The content of the placebo was just maize flour in the similar doses as provided the jaman seed i.e. 1, 3, and 6 gm per day per individuals
The values without superscripts with the rows are not statistically significant.

3 = 66 placebo (Maike Flora) fed to the diabetic individuals per day.

2 = 36 placebo (Maike Flora) fed to the diabetic individuals per day.

1 = 16 placebo (Maike Flora) fed to the diabetic individuals per day.

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Table 9: Effect of Placebo on Blood Cholesterol in Diabetic Individuals
7. EFFECT OF JAMAN ON HIGH-DENSITY LIPOPROTEIN (HDL) OF DIABETIC INDIVIDUALS

7.1 RESPONSE OF HDL OF DIABETIC INDIVIDUALS TO JAMAN FRUITS

The effects of different doses of Jaman fruit on fasting blood high-density lipoprotein (HDL) levels of diabetic individuals were non-significantly different.

The mean values of the fasting blood HDL of groups-1 (6 Jaman per day per individual), group-2 (12 Jaman per day per individual) and group-3 (18 Jaman per day per individual) are presented in Table-10.

Before the intake of jaman fruit, the mean fasting blood HDL of diabetic individuals of group-1, was 32.0 mg/dl which reduced 8.12+3.4% on day 10 and then increased 1.8+1.3% on day 20. The HDL values again reduced 6.8+3.4% on day 30 and an increased 5.9+2.3% was recorded on day 40. In group-2 data was 34.7 mg/dl which increased 0.8+1.0%, 1.7+3.2, 0.8+3.4, and 1.1+0.3% after 10, 20, 30 and 40 days. In group-3 diabetic individuals fasting blood HDL was observed 36.8 mg/dl which increased 1.8+0.4%, 4.6+7.8, 5.9+8.3, 5.9+6.6% after 10, 20, 30, and 40 days (Appendix-VII).
The values without superscripts with the rows are not statistically significant.

3* = 18Jamnu fruit led to the diabetic individuals per day.
2* = 12Jamnu fruit led to the diabetic individuals per day.
1* = 6 Jamnu fruit led to the diabetic individuals per day.

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**Table 1**: Effect of different doses of Jamnu fruit on blood HDL in diabetic individuals.
The mean fasting blood HDL values of diabetic individuals of group-1 on day 0 was 38.8 and 25.1 mg/dl for male and female respectively (Table-10). After the intake of jaman fruit (6 jaman per day) for 10 days, the mean fasting blood HDL levels reduced 35.0 and 24.8 mg/dl (18.3 to 0.9%) for male and female respectively. With further intake of jaman fruit for 10 days, the blood HDL of the same individuals increased to 39.6 and 25.6 mg/dl (2.0 to 1.9%). Further intake for 10 days, Jaman fruits reduced the mean fasting blood HDL 33.3 mg/dl in male while in female the mean fasting HDL values were unchanged 25.6 mg/dl on days 30. After 10 day of the termination of Jaman fruit intake (recorded on 40th day) the mean fasting blood HDL of both male and female increased to 39.8 to 28.0 mg/dl (3.8 to 11.5%) in comparison to the data recorded during the jaman intake.

The mean fasting blood HDL values of group-2 individuals on day 0 was 37.6 to 31.8 mg/dl for male and female respectively (Table-10). After the intake of jaman fruit (6 jaman per day) for 10 days, the mean fasting blood HDL levels in male diabetic individual were slightly changed 37.1 mg/dl (1.3%) while the HDL values of female diabetic individuals increased 33.0 mg/dl (2.2%) respectively. With further intake of jaman fruit for 10 days, the blood HDL of the male diabetic individuals increased 38.1(1.0%) while in female decreased 32.5 (0.9%). Further intake for 10 days, Jaman fruits showed the same effect on blood HDL levels in diabetic individuals. After 10 day of the termination of Jaman fruit intake (recorded on 40th day) the mean fasting blood HDL of male diabetic individuals increased 38.6 mg/dl (2.6%) but no increased in HDL was observed in female diabetic individuals 31.6 mg/dl (0.6%)
The mean fasting blood HDL values of group-3, diabetic individuals on day 0 was 41.5 to 32.2 mg/dl for male and female respectively (Table-10). After the intake of jaman fruit (18 no per day) for 10 days, the mean fasting blood HDL levels in male diabetic individual slightly reduced 39.2 (5.5%) but the HDL mean values on day 10 of female increased 33.0 mg/dl (2.4%) respectively. With further intake of jaman fruit for 10 days, the blood HDL of the male diabetic individuals remained unchanged 39.2 mg/dl (5.5%) but the mean HDL values of female reduced to 31.0 (3.7%). Further intake for 10 days, Jaman fruits showed the same effect on blood HDL levels in diabetic individuals. After 10 day of the termination of Jaman fruit intake (recorded on 40th day) the mean fasting blood HDL of male diabetic individuals were noted lower 38.0 (5.9%) as compared with the values on day 0. Similarly the mean fasting HDL values of female 31.2 (3.1%) is lower than the values on day 0 but similar to the values recorded during the jaman intake.

There was inconsistent effect of the various jaman fruits doses on the HDL-cholesterol concentration of the diabetic subjects (Table-10). There are no reports of jaman on the HDL cholesterol in the literature. However, this finding is not in agreement with other previous reports in other medicinal plants (Anita et al. 2002).

Khan et al (1996) found that curry leaf (Murraya Koenigii) and mustard seeds (Brassica Juice) also increased significantly HDL levels. Further, Khan et al (1998) claimed that spices- Murraya Koenigii and Brassica juncea reduced the concentrations of cholesterol, TGL and phospholipids in serum, aorta, liver and
heart. The low-density lipoproteins and very low-density lipoprotein fractions were also decreased and the HDL fraction was increased. Mani et al (2000) reported that spirulina tablet significantly reduced serum cholesterol and free fatty acid in diabetic individuals.

However, supplementation of buckwheat, butcher's broom and hydroxyethyl rutsid have significantly decrease diabetic retionopathy in humans and improved local circulation in the retina. They also lowered cholesterol, triglyceride and raised HDL cholesterol. Cinnamon also contained procyanidian dimers and oligomers that improve capillary function (Archimowicz et al (1996) and (Morimoto et al 1986). In another study Arun et al (1981) reported that garlic decreased serum cholesterol, triglycerides, low-density liprotein and increased the high-density protein.

7.2 RESPONSE OF HDL OF DIABETIC INDIVIDUALS TO JAMAN SEED

The mean fasting blood HDL values of the selected diabetic individuals of group-1 was 35.0±6.6 mg/dl which increased 0.0±0.0%, 0.0±0.0, 1.4±1.3, 4.5±1.4% after 10, 20, 30 and 40 days. In group-2 the mean fasting HDL values 20.0±14.2 mg/dl which increased 27.8±4.2%, 30.7±10.4, 32.1±8, 16.7±14.4% after 10, 20, 30, and 40 days. In group-3 the mean fasting HDL was 27.7±2.2 mg/dl which increased 5.3±13.4%, 8.6±14.9, 13.7±10.4,13.7±12.1% after 10, 20,30, and 40 days. (Appendix-VIII)
The values without superscripts with the rows are not statistically significant.

3 = 68 lambs fed to the diabetic individuals per day.

2 = 38 lambs fed to the diabetic individuals per day.

1 = 18 lambs fed to the diabetic individuals per day.

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</tr>
<tr>
<td>32.8±1.3</td>
<td>30.5±1.3</td>
</tr>
<tr>
<td>31.7±2.9</td>
<td>33.5±2.6</td>
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<tr>
<td>36.8±3.5</td>
<td>34.8±2.6</td>
</tr>
<tr>
<td>37.3±4.2</td>
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</tr>
<tr>
<td>35.8±6.0</td>
<td>33.5±6.3</td>
</tr>
</tbody>
</table>

Table 11: Effect of different doses of lambs on blood HDL in diabetic individuals.
The mean fasting blood HDL values of the selected diabetic individuals of group-1 was 34.8 and 34.8 mg/dl for male and female individuals were recorded on day 0 of the trial respectively (Table-11). After the intake of jaman seed (1 gm per day per individual) for 10 days, the mean fasting blood HDL levels in male diabetic individuals were increased to 35.7 mg/dl (8.8%) but in female the mean values reduced 34.3 mg/dl (0.4%).

With further intake of jaman fruit for 10 days, the blood HDL of the same individuals slightly reduced 35.5 mg/dl (9.2%) but in female the values increased 34.7 mg/dl (12.6%) respectively. Again with further intake of jaman seed for 10 days the HDL values of male diabetic individuals increased 36.3 mg/dl (8.7%) but the mean values of HDL in female 34.7 mg/dl (2.4%) unchanged with the intake of jaman seed. After 10 days of discontinuation of the jaman seed dose the increased of 35.8 mg/dl (9.2%) and 37.3 mg/dl (0.04%) was recorded on day 40 of the trial. This indicated that prolong use of the jaman seed improved the HDL level in diabetic individuals (Table-11).

The mean fasting blood HDL values of the selected diabetic individuals of group-2 was 37.7 and 34.0 mg/dl for male and female individuals were initially recorded on day 0 of the trial respectively (Table-11). After the intake of jaman seed (3 gm per day per individual) for 10 days, the mean fasting blood HDL levels of male diabetic individuals were reduced 37.5 mg/dl (0.5%) in female diabetic individuals. While the mean HDL values increased 35.3 mg/dl (3.5%). The same dose of jaman seed were regularly taken by the diabetic individuals.
for further 10 days the HDL levels of the same individuals slightly increased 37.8 and 35.5 mg/dl (0.2 to 4.4%). With further intake for more 10 days the mean values of HDL in male and female again slightly reduced 34.3 and 31.8 mg/dl (9.0 to 6.4%) in both sex respectively.

After day 30 of the feeding trial the dose of jaman seed were discontinued and blood of these individuals were taken on day 40 to find the after effect of the jaman on HDL levels. The mean value was 36.7 and 31.5 mg/dl (2.6 to 7.3%) on day 40 but lower than day 0 but higher than day 30.

After day 30 the intake of the dose were discontinued by the individuals and the HDL values in male and female increased 36.7 and 31.5 mg/dl (1.7 to 3.0%) respectively. This indicated that jaman seed had a longer effect in the increased of HDL levels in diabetic individuals.

The HDL values of the selected diabetic individuals of group-3 was 28.0 and 27.5 mg/dl for male and female individuals were recorded on day 0 of the trial (Table-11). After the intake of jaman seed (6 gm per day per individual) for 10 days, the mean fasting blood HDL levels of male and female diabetic individuals were increased 28.5 and 30.0 mg/dl (1.2 to 9.0%) in male and female individuals. The individuals continued the same dose for 10 days further the HDL levels increased 29.8 and 30.5 mg/dl (3.5 to 10.9%) in male and female individuals. The same individuals used the same for more 10 days the HDL values gradually increased 30.5 and 31.8 mg/dl (6.4 to 15.9%) on day 30 of the experiment. After 10 days discontinuation of the dose (jaman seed) the mean
HDL level increased 32.8 to 30.0 mg/dl (8.9 to 9.0%) were observed on day 40 of the experiment. This indicated that jaman seed have prolonged and regular effect in the increased of HDL levels in diabetic individuals (Table 11).

Our results indicated inconsistent increased in HDL levels in group-1, while in group-2 and group-3 HDL levels were consistently increased in diabetic individuals. The finding of this study are similar with those of Anita et al (2002). They reported that bittergourd (karela), jaman, and fenugreek seed (methi seeds) significantly improved the serum lipid profile, lower total, LDL cholesterol, VLDL cholesterol and triglycerides and increased HDL cholesterol level. Khan et al (1996) found that curry leaf (Murraya Koenigii) and mustard seeds (Brassica Juice) fed to rats and that increased HDL levels significantly. Khan et al (1998) also reported that spices- Murraya Koenigii and Brassica juncea reduced the concentrations of cholesterol, TGL and phophholipids in serum, aorta, liver and heart. The low-density lipoproteins and very low-density lipoprotein fractions were also decreased and the HDL fraction was increased. However, supplementation of buckwheat, butcher’s broom and hydroxyethyl rutside significantly decrease diabetic retinopathy in humans and improve local circulation in the retina. They also lowered cholesterol and triglyceride and raised HDL cholesterol. Arun et al (1981) reported that garlic significantly lowered serum cholesterol and triglycerides but increase the high-density lipoproteins.
7.3 EFFECT OF PLACEBO ON BLOOD HDL LEVEL IN DIABETIC INDIVIDUALS

As mentioned earlier, these experiments were conducted on diabetic individuals and were self control. However, placebo trials were conducted in similar pattern in order to see the psychological effect of the jaman seed. The content of the placebo was just maize flour in the similar doses as provided the jaman seed i.e. 1, 3, and 6 gm per day per individuals.
The values without superscripts with the rows are not significantly different.

$3^* = 6\%$ Placebo (Maiasa Flour) fed to the diabetic individuals per day.

$2^* = 3\%$ Placebo (Maiasa Flour) fed to the diabetic individuals per day.

$1^* = 1\%$ Placebo (Maiasa Flour) fed to the diabetic individuals per day.

<table>
<thead>
<tr>
<th>Day</th>
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<th>2</th>
<th>3</th>
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<th>5</th>
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<th>7</th>
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<th>10</th>
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<td>5</td>
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</tr>
<tr>
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</tr>
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<td>Mean ± SD</td>
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</tr>
<tr>
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<td><strong>Doses of Placebo (mg/dl)</strong></td>
<td><strong>Group</strong></td>
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<td></td>
</tr>
</tbody>
</table>

**Table 12**: Effect of Placebo on Blood HDL Level in Diabetic Individuals

**Fasting Blood HDL (mg/dl)**
8. **EFFECT OF JAMAN ON LOW-DENSITY LIPOPROTEIN OF DIABETIC INDIVIDUALS**

8.1 **RESPONSE OF LDL OF DIABETIC INDIVIDUALS TO JAMAN FRUITS**

The mean fasting blood LDL values of the selected diabetic individuals of group-1, was 148.5 mg/dl which increased 4.5±2.5%, and reduced 0.4±2.1, 2.9±4.2 and 2.2±2.5 after day 10, 20, 30, and 40 days. In group-2 data was 136.5 mg/dl which reduced 2.4±9.6%, 0.17±0.2, 0.04±4.3, 0.5±1.2 of day 40. While in group-3 the mean fasting blood LDL level of the individuals was 147.7 mg/dl which show no increased 0.0±0.0, on day 10 but the LDL levels increased 1.0±2.5, 2.6±2.9, 2.7±4.0 after 20, 30, and 40 days (Appendix-IX).

8.2 **EFFECT OF JAMAN FRUIT ON LDL IN MALE AND FEMALE DIABETIC INDIVIDUALS**

The mean fasting blood LDL values of the selected diabetic individuals of group-1 on day 0 was 147.1 and 150.0 mg/dl for male and female respectively (Table-13). After the intake of jaman fruit (6 jaman per day per individuals) for 10 days, the mean fasting blood LDL levels increased 157.8 and 153.1 mg/dl (7.2 to 2.06%) in male diabetic individuals. Similarly, the mean fasting values of LDL on day 20 was again reduced 145.2 mg/dl (1.29%) in male and 150.5 mg/dl (0.33%) in female respectively. With further intake of jaman fruit for 10 days, the blood LDL of the same individuals reduced 144.0 and 146.8 mg/dl (2.10 to
The values without superscripts with the rows are not statistically significant.

<table>
<thead>
<tr>
<th>Dose groups</th>
<th>Effect of different doses of Jamun Fruit on Blood LDL in Diabetic Individuals</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>18 Jamun Fruit fed to the diabetic individuals per day</td>
</tr>
<tr>
<td>2</td>
<td>12 Jamun Fruit fed to the diabetic individuals per day</td>
</tr>
<tr>
<td>3</td>
<td>6 Jamun Fruit fed to the diabetic individuals per day</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Day 0</th>
<th>Mean ± SD</th>
<th>Mean ± SD</th>
<th>Mean ± SD</th>
<th>Mean ± SD</th>
</tr>
</thead>
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<td>158.3 ± 37.0</td>
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<td>0</td>
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<td>143.0 ± 21.5</td>
</tr>
<tr>
<td>0</td>
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<td>137.6 ± 14.2</td>
</tr>
<tr>
<td>0</td>
<td>146.8 ± 5.9</td>
<td>150.0 ± 9.6</td>
<td>150.5 ± 9.7</td>
<td>151.0 ± 9.8</td>
</tr>
<tr>
<td>0</td>
<td>143.5 ± 34.7</td>
<td>145.2 ± 41.9</td>
<td>147.8 ± 49.9</td>
<td>147.1 ± 56.4</td>
</tr>
</tbody>
</table>

Table 13: Effect of different doses of Jamun Fruit on Blood LDL in Diabetic Individuals.
The mean fasting blood LDL values of the selected diabetic individuals of group-1 on day 0 was 147.1 and 150.0 mg/dl for male and female respectively (Table-13). After the intake of jaman fruit (6 jaman per day per individuals) for 10 days, the mean fasting blood LDL levels increased 157.8 and 153.1 mg/dl (7.2 to 2.06%) in male diabetic individuals. Similarly, the mean fasting values of LDL on day 20 was again reduced 145.2 mg/dl (1.29%) in male and 150.5 mg/dl (0.33%) in female respectively. With further intake of jaman fruit for 10 days, the blood LDL of the same individuals reduced 144.0 and 146.8 mg/dl (2.10 to 2.13%). Further intake for 10 days, Jaman fruits reduced the mean fasting blood LDL 143.5 mg/dl (2.4%) but the values of female again increased 148.0 mg/dl (1.3%). After the termination of jaman fruit for 10 days the mean fasting blood LDL levels of male individuals reduced as compared to day 30 and day 0 (control 0). The mean values of LDL of female diabetic individuals on day 40 was increased (1.3%) as compared to day 30 and day 0 (control 0).

The mean fasting blood LDL values of the selected diabetic individuals of group-2 on day 0 was 138.0 to 135.0 mg/dl for male and female respectively (Table-13). After the intake of jaman fruit (12 jaman per day) for 10 days, the mean fasting blood LDL levels reduced 136.3 mg/dl (5.7%) in male diabetic individuals. While the mean fasting values of LDL in female diabetic individuals was increased 136.3 mg/dl (0.96%) respectively. With further intake of jaman fruit for 10 days, the blood LDL of the same individuals reduced 137.6 to 135.0 mg/dl (0.28 to 0.0%). Further intake for 10 days, Jaman fruits reduced the mean fasting blood LDL 136.1 mg/dl (1.37%) but the values of female again increased
136.3 mg/dl (0.96%). After the termination of jaman fruit for 10 days the mean fasting blood LDL levels of male individuals increased as compared to day 30. While the mean values of LDL of female on day 40 no increased were recorded by comparing to day 0 (Control).

The mean fasting blood LDL values of the selected individuals of group-3 on day 0 was 137.2 to 158.3 mg/dl for male and female respectively (Table-13). After the intake of jaman fruit (12 jaman per day) for 10 days, the mean fasting blood LDL levels increased 138.7 mg/dl (1.09%) in male diabetic individuals. While In female, the mean fasting values of LDL reduced 156.8 mg/dl (0.75%) on day 10. Similarly the mean fasting LDL values of male were consistently increased 139.2, 142.0, and 144.0 mg/dl (1.4, 3.4 4.9%) on 20, 30 and 40 days respectively.

The LDL values of female individuals were also reduced 156.8 mg/dl on day 10, after intake of jaman fruit and than consistently increased 159.5, 161.3 and 159.7 mg/dl (0.7,1.8,0.88%) respectively.

Our results indicated that LDL was non- significantly reduced in the jaman fruit doses group-1,2 and 3. However, higher % reduction was recorded in group-1 on day 10 as compared with other doses. While in group-2 and 3 LDL level was reduced consistently with the increased of duration of jaman intake. Anita et al (2002) administered bittergourd (karela), jaman, and fenugreek seed (methi seeds), and a significant improvement in the serum lipid profile and lowering total, LDL, cholesterol, VLDL cholesterol. Murraya
Koenigii and Brassica juncea highly reduced the cholesterol and low-density lipoproteins and very low-density lipoprotein fractions were also reduced. These herbs help in controlling of hypercholesterolaemia and arteriosclerosis development in diabetic individuals (Khan et al., 1998).

Further, Khan et al. (1996) reported that curry leaf (Murraya Koenigii) and mustard seeds (Brassica Juice), reduced total serum cholesterol and LDL and VLDL.

8.3 **RESPONSE OF LDL OF DIABETIC INDIVIDUALS TO JAMAN SEED**

The mean fasting blood LDL values of the selected diabetic individuals of group-1 was 157.6±19 mg/dl on day 0, which reduced 0.5±2.1%, 2.3, 2.8, 1.5% after 10, 20, 30, and 40 days. In group-2 the mean fasting LDL was 168.7±28.0 mg/dl on day 0, which reduced 2.8±3.5%, 1.5±3.5% on day 10 and 20 but increased 4.5±8.1 and 1.3±8.5% on day 30 and 40 days in male and female respectively (Appendix-IX). While in group-3 blood LDL was 152.10 ±8.0 mg/dl on day 0, which reduced 1.5±17.7%, 16.0±28.0, 5.9±29.3% and increased in LDL values was noted on 40 days, after 10, 20, 30 days (Appendix-IX).
The values without superscripts with the rows are not statistically significant.

3* = 6g Jamman seed fed to the diabetic individuals per day.
2* = 2g Jamman seed fed to the diabetic individuals per day.
1* = 1g Jamman seed fed to the diabetic individuals per day.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Day 30</th>
<th>Day 20</th>
<th>Day 10</th>
<th>Mean ± SD</th>
<th>Mean ± SD</th>
<th>Mean ± SD</th>
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<th>6</th>
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</thead>
<tbody>
<tr>
<td>153.3 ± 30.7</td>
<td>143.8 ± 25.3</td>
<td>113.3 ± 22.5</td>
<td>101 ± 20.5</td>
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<tr>
<td>120.0 ± 50.5</td>
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<td>10.5 ± 11.7</td>
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<td>14.2 ± 11.7</td>
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<td>1.0 ± 0.1</td>
<td>1.0 ± 0.1</td>
</tr>
<tr>
<td>139.7 ± 15.0</td>
<td>14.2 ± 11.7</td>
<td>134.8 ± 14.1</td>
<td>14.2 ± 11.7</td>
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<td>1.1 ± 0.1</td>
<td>1.0 ± 0.1</td>
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</tr>
</tbody>
</table>

Table 1.4: Effect of different doses of Jamman seed on blood LDL in diabetic individuals.
The mean fasting blood LDL values of the selected diabetic individuals of group-1 on day 0 was 109.2 and 138.2 mg/dl for male and female individuals respectively (Table-14). After the intake of jaman seed (1gm per day per individual) for 10 days, the mean fasting blood LDL levels reduced 108.5 and 137.7 mg/dl (0.64 to 0.36%) in male and female diabetic individuals respectively. With further intake of jaman seed for 10 days, the blood LDL of the same individuals reduced to 106.7 and 134.3 mg/dl (2.2 to 2.8%) in both sexes. With more 10 days intake of jaman seed the mean reduction in LDL was 105.2 and 134.7 mg/dl (3.6 to 2.5%) respectively. After termination of this dose for 10 days the mean fasting LDL values was 107.2 and 136.7 mg/dl (1.8 to 1.08%) was recorded. These values comparatively lower than day 0 as well from the mean values of the feeding trial.

Regarding the mean fasting LDL values of the selected diabetic individuals of group-2 on day 0 was 84.0 and 145.3 mg/dl for male and female individuals respectively (Table-14). After the intake of jaman seed (3gm per day per individual) for 10 days the mean fasting LDL levels reduced 83.5 and 138.8 mg/dl (0.5 to 4.4%) in male and female diabetic individuals respectively. With further intake of jaman seed for 10 days, the blood LDL of the male diabetic individuals reduced 83.3 (0.8%) but the mean LDL values of female increased 142.5 mg/dl (1.9%). Further intake for 10 days the mean fasting blood LDL level was increased 91.0 and 144.7 mg/dl (8.3 to 0.4%), in male and female diabetic individuals. After the termination of jaman seed doses for 10 days the mean fasting blood LDL levels on day 40 of male individuals remain high 89.0 and
139.7 mg/dl (5.9 to 3.8%) as compared to the values on day 30 and day 0 (control).

In group-3 the mean fasting LDL values of the selected diabetic individuals of on day 0 was 135.0 and 131.5 mg/dl for male and female individuals respectively (Table-14). After the intake of jaman seed (6gm per day per individual) for 10 days, the mean fasting LDL levels reduced 121.5 and 101.0 mg/dl (10.0 to 23.1%) in male and female diabetic individuals respectively. With further intake of jaman seed for 10 days, the blood LDL of the same individuals reduced 105.5 and 113.5 mg/dl (21.8 to 13.8%). Further intake for 10 days increased the mean fasting LDL in male and female diabetic individuals. After the termination of jaman seed doses for 10 days the mean fasting blood LDL levels on day 40 of male individuals remain low 120.0 mg/dl (11.1%) as compared to day 0 (Control). The LDL value of female on day 40 was 153.2 mg/dl (16.5%) as compared to the values on day 30 and day 0.

Our results showed that jaman seed doses (1, 2 and 3gm) non significantly reduced LDL in diabetic individuals. The % reduction in LDL of group-2 and group-3 was higher as compared with the group-1. Our finding are in line with the earlier reported (Anita et al. 2002) and (Khan et al. 1998). In another study Khan et al (1996) tested curry leaf (Murraya Koenigii) and mustard seeds (Brassica Juice) reduced total serum cholesterol, LDL and VLDL.
9. **EFFECT OF PLACEBO ON LOW DENSITY LIPOPROTEIN (LDL) IN DIABETIC INDIVIDUALS**

As mentioned earlier, these experiment were conducted on the diabetic individuals and were self control. However, placebo trial were conducted in similar pattern in order to see the psychological effect of the jaman seed. The content of the placebo was just maize flour in the similar doses as provided the jaman seed i.e. 1, 3, and 6 gm per day per individuals.
The values without superscripts with the rows are not statistically significant.

<table>
<thead>
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<th>Dose of Placebo (mg/day)</th>
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<th>Day 20</th>
<th>Day 30</th>
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<th>After Intervention</th>
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<td>137.0 ± 19.9</td>
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<td>115.3 ± 19.7</td>
<td>130.7 ± 19.8</td>
<td>134.6 ± 19.2</td>
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<td>148.1 ± 19.9</td>
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<td>133.6 ± 19.9</td>
<td>115.3 ± 19.7</td>
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<td>133.6 ± 19.9</td>
<td>115.3 ± 19.7</td>
<td>130.7 ± 19.8</td>
<td>134.6 ± 19.2</td>
</tr>
</tbody>
</table>

Table 15: Effect of Placebo on Blood LDL Level in Diabetic Individuals

3* = 6g Placebo (Maize Flour) Fed to the diabetic individuals per day.
2* = 3g Placebo (Maize Flour) Fed to the diabetic individuals per day.
1* = 1g Placebo (Maize Flour) Fed to the diabetic individuals per day.
10. **MICRONUTRIENTS CONCENTRATION IN JAMAN FRUITS & SEEDS**

The data regarding micronutrient status of Jaman fruit are presented in Table-15. The results indicated that Zn content of Jaman fruit is higher which is 0.13 mg kg$^{-1}$ as compared to jaman seed (0.068 mg kg$^{-1}$).
<table>
<thead>
<tr>
<th>Micronutrient</th>
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<th>Jamun Seed</th>
</tr>
</thead>
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<td>Cadmium</td>
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</tr>
<tr>
<td>Nickel</td>
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<td>Copper</td>
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<td>Platinum</td>
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</tr>
<tr>
<td>Manganese</td>
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<tr>
<td>Iron</td>
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<tr>
<td>Chromium</td>
<td>0.013</td>
<td>0.0068</td>
</tr>
</tbody>
</table>

Table 16: Micronutrient Concentration in Jamun Fruit & seed per 100 Grams.
As far as the concentration of chromium in fruit is concerned, it was found as 6.00 mg/kg, which is also lowered from that of seed. The other minerals like iron, Manganese, Plumbum, copper, Nickel and cadmium content of Jaman fruit were 3.92, 0.242, 11.8, 0.03, 0.158 and 0.114 mg/kg respectively, while that of seed were 0.82, 0.224, 8.74, 1.292, 0.216, and 0.024 respectively. The results are in fair agreement with those of Nooral (1996) who determined the minerals contents of jaman fruits.

Our result indicated that jaman fruit and jaman seed reduced the mean fasting blood glucose in Type-II diabetic individuals. These natural plant products are a good source of micronutrients, specially zinc, chromium and iron. The reduction of fasting blood glucose in diabetic individuals may be due to the presence of these micronutrients. Mertz (1969) reported that chromium is an essential part of diet that improve glucose tolerance and chromium deficient rates.
5. SUMMARY

Diabetes is a chronic degenerative disease caused by a lack of or resistance to insulin, which is essential for the proper metabolism of blood sugar. Diabetes mellitus is possibly the world’s fastest growing disease and continues to be a threat to public health around the world. Since long before the insulin period, indigenous drugs have been in used for the treatment of this disease. The traditional plants drugs represent a mild form of therapy with fewer drawbacks than chemical either isolated from vegetable kingdom or synthesized. Pharmacological studies have been showed usefulness of plant extracts in diabetes, although they do not indicate adequate effectiveness of these hypoglycemic agents (unknown). A hypoglycemic action of Jaman (Eugenia jambolana or Syzygium cumini) has been confirmed in animal model. Therefore, a study was conducted on human volunteers to explore the possible hypoglycemic action in human. Fifty four male and fifty four female diabetic individuals were selected who were 40-65 years of age having blood glucose and triglyceride in the of rang of 140 to 250 and 200 to 250 mg/dl. The diabetic individuals were divided into 3 major groups i.e. Fruit, Seed and Placebo. Each group was further sub-divided into 3 groups (for doses). Each sub-group contained 6 male and 6 females. For jaman fruit group individuals in group-1 were given 6 jamans, group-2, 12 jamans and group-3, 18 jamans. For jaman seed group-1 was given 1g, group-2, 3 gm and group-3, was given 6gm. All the trials were conducted for 30 days and after termination of the dose the same individuals were observed for 10 days. The doses of jaman, seed and placebo
were given with breakfast, lunch and dinner. Data was recorded on days 0, 10, 20, 30 and 40 for glucose, TGL, cholesterol, HDL, and LDL. Jaman fruit induced consistently hypoglycemic effect on blood glucose of all the individuals. The % reduction in the mean fasting glucose (excluding the sex factor) of group-1, diabetic individuals (6 Jaman) was 188.6 mg/dl which reduced 1.2±1.1%, 2.7±3.0, 4.0±4.4, 1.5±1.7% after 10, 20, 30, and 40 days. In group-2 (12 Jaman) data was 228.5±17.8 mg/dl which reduced 1.0±5.1%, 1.8±6.2, 3.4±6.5, and 1.8±5.7% after 10, 20, 30 and 40 days. In group-3 (18 Jaman) blood glucose was recorded as 223.3±23.6 mg/dl which reduced 0.5±4.3%, 2.1±4.0, 3.6±5.4 and 1.7±4.1% after 10, 20, 30 and 40 days. The doses of jaman non-significantly reduced the blood glucose levels in diabetic individuals. The % reduction in the mean fasting blood glucose (excluding the sex factor) of group-1, individuals (1gm jaman seed) was 227.8 mg/dl which reduced 0.2±1.2%, 1.0±2.1, 1.7±0.2, and 2.0±1.0% after 10, 20, 30, and 40 days. In group-2 (3 gm Jaman seed) data was 168.7 mg/dl which reduced 5.1±2.1%, 3.0±0.4, 3.0±2.0, and 3.0±2.0% after 10, 20, 30 and 40 days. In group-3 (6 gm Jaman seed) blood glucose was recorded as 157.10 mg/dl which reduced 2.7±0.4%, 2.1±1.1, 2.1±0.5 and 2.6±2.1% after 10, 20, 30 and 40 days. The % reduction in the mean fasting blood TGL (excluding the sex factors) group-1, (6 Jaman) was 225.9 mg/dl which reduced 0.8±2.0%, 1.9±4.1, 2.8±1.2, and 0.2±1.9% after 10, 20, 30, and 40 days. In group-2 (12 jaman) data TGL was 227.5 mg/dl which reduced 0.6±1.1%, 0.3±3.0, 2.1±4.4, and 0.13% after 10, 20 and 30 and 40 days. In group-3 (18 Jaman) blood TGL was recorded as 230.3 mg/dl which reduced 3.5±11.3%, 5.3±11.3, 6.6±10.4,
4.6±12.6% after 10, 20, 30 and 40 days. The % reduction in the mean fasting group-2 data was 229.9 mg/dl which increased 0.6±1.1%, 0.3±3.0, 2.1 ± 4.4, 0.13% in all the individuals. In group-3, the fasting TGL level was 236.3, mg/dl which increased 3.5, 5.3 and 6.6% in diabetic individuals. However, % reduction in the mean fasting blood cholesterol levels of group-1 (6 Jaman) was 225.6 mg/dl which reduced 4.2±4.1%, 7.3±4.8, 10.2±4.9 and 5.8±2.8 after 10, 20, 30, and 40 days. In group-2 data was 207.4 mg/dl which reduced 2.2±5.8%, 4.5±5.7, 6.8±6.3 and 4.9±4.2±9.1% after 10, 20, 30 and 40 days. The data of group-3, diabetic individuals showed the mean fasting blood cholesterol was 208.2 mg/dl, which reduced 3.4±3.2%, 6.5 ±4.7, 9.6±5.1 and 6.0±5.0 % after 10, 20,30, and 40 days. The % reduction in the fasting blood cholesterol (excluding the sex factor) of group-1, was 205.3±7.9 mg/dl which reduced 3.9±4.1%, 4.6±4.8, 4.5±4.9 and 3.4±2.8 after 10, 20, 30, and 40 days. In group-2 the mean fasting values of cholesterol was 197.3±35.0 mg/dl which reduced 1.5±1.1, 0.6±5.7% on 10 and 20 days but an increased of 0.5±8.8% was recorded on 30 days again 1.3±3.4% reduced on day 40. In group-3, the mean fasting cholesterol of the diabetic individuals was 213.0±26.3 mg/dl which reduced 11.6±14.8%, 12.2±17.2, 10.7±19.7% after 10, 20, 30 days and an increased of 4.7±23.3 % after 40 days was recorded. The mean fasting HDL level of group-1, (jaman fruit) diabetic individuals was 32.0 mg/dl which reduced 8.12±3.4% on day 10 and than increased 1.8±1.3% on day 20. The HDL levels
again reduced 6.8±3.4% on day 30 and an increased 5.9 was recorded on day 40. In group-2 data was 34.7 mg/dl which increased 0.8±1.0%, 1.7±3.2, 0.8±3.4, and 1.1±0.3% after 10, 20, 30 and 40 days. In group-3 diabetic individuals fasting HDL was observed 36.8 mg/dl which increased 1.8±0.4%, 4.6±7.8, 5.9±8.3, 5.9±6.6% after 10, 20, 30, and 40 days. The mean fasting blood HDL levels of group-1, (jaman seed) diabetic individuals was 35.0±6.6 mg/dl and no changed was observed on day 10, 20 and than an increased of 1.4±1.3, 4.5±1.4% after 30 and 40 days was recorded. In group-2 the mean fasting HDL levels was 20.0±14.2 mg/dl which increased 27.8±4.2%, 30.7±10.4, 32.1±8, 16.7±14.4% after 10, 20, 30, and 40 days. In group-3 the mean fasting HDL was 27.7±2.2 mg/dl which increased 5.3±13.4%, 8.6±14.9, 13.7±10.4, 13.7±12.1% after 10, 20, 30, and 40 days., The mean fasting blood LDL levels of group-1, diabetic individuals was 148.5 mg/dl which increased 4.5±2.5%, on day 10 and than reduced 0.4±2.1, 2.9±4.2 and 2.2±2.5% after 20, 30, and 40 days. In group-2 data was 136.5 mg/dl which reduced 2.4±9.6%, 0.17±0.2, 0.04±4.3 and 0.5±1.2% on day 40. While in group-3 the mean fasting blood LDL level was 147.7 mg/dl which showed an increased of 1.0±2.5, 2.6±2.9, 2.7±4.0% after 20, 30, and 40 days. In micronutrients, jaman fruit contain 0.13 Zn, 0.034 Cu, 11.88 Pb, 6.00 Cr, 3.92 Iron, 0.242 Mn, 0.158 Ni, 0.114 Cd while jaman seed also contain 0.0068 Zn, 1.292 Cu, 8.74 Pb, 14.00 Cr, 0.82 Iron 0.224 Mn, 0.216 Ni and 0.114 Cd mg/100 g which are somehow related with the diabetes.
6. LIMITATION OF THE STUDY

This project was difficult in the execution since it involve human subjects. The availability of the subjects, the willingness and motivation for the informed consent was one of the difficult processes. Most of the patient (which were not involved) hesitated from blood sampling for some reason. The age limit, criteria sit for the ranges of blood glucose and triglyceride were also obstacle for the experiment. A large numbers of diabetics had screened in order to get the desirable age and blood glucose & triglyceride ranges. Another limitation was female subjects who were not only difficult to be controlled but were also difficult to manage due to norms of society.

The funding was very limited for this research project and most of the expenses were born by the scholar. The transport facilities were also limited in this study.

The dietary control was subjected to their usual food habits. These subjects were not restricted and standardized for diets due to ethical reasons. Therefore there must be confounding factors which might have come during the experiment.

Since this was a self controlled study and there were no restrictions on the exercise or dietary intake.
7. RECOMMENDATIONS

The present study indicates that jaman fruits and seed have positive effects on the reduction of hyperglycemia in the diabetic individuals. It is recommended that the inclusion of either jaman fruits or seed would be beneficial if included in the routine intake. It is further recommended that the increased duration of intakes would be more useful for better results. The jaman fruit is short season fruit therefore it must be preserved in order to insure its availability throughout the year for diabetic individuals. Further research is needed to investigate the effect of jaman fruit and seed on the blood glucose and lipid of the diabetic individuals at molecular level.
8. LITERATURE CITED


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Virmani, P., Gupta,S., Misa, P.(2001): Effect of Syzygium Cumini (Jaman) Supplementation on blood glucose and lipid profile in NIDDM. Institute of Home Economics (Delhi University) and Department of Medicine. Department of biostatistics, All India Institute of Medical Sciences, New Delhi. J. of Ethnopharmacology. 30: 211-214.


### APPENDICES

**Appendix-I: Effect of Jaman fruit on blood glucose in diabetic individuals (excluding the sex factor)**

<table>
<thead>
<tr>
<th>Doses groups</th>
<th>Sex</th>
<th>Fasting Blood Glucose mg/dl</th>
</tr>
</thead>
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<tr>
<td>2*</td>
<td>M+F</td>
<td>228.5±17.8</td>
</tr>
<tr>
<td>3*</td>
<td>M+F</td>
<td>223.3±23.6</td>
</tr>
</tbody>
</table>

1* = Indicate 6 Nos. of Jaman fruit fed to the diabetic individuals.
2* = Indicate 12 Nos. of Jaman fruit fed to the diabetic individuals.
3* = Indicate 18 Nos. of Jaman fruit fed to the diabetic individuals.
Appendix-II: Effect of Jaman seed on blood glucose in diabetic individuals (excluding the sex factor).

<table>
<thead>
<tr>
<th>Groups of Diabetics</th>
<th>Sex</th>
<th>Fasting Blood Glucose mg/dl</th>
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<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Before Intervention</td>
<td>During Intervention</td>
<td>After Intervention</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Day 0 Day 10 Day 20 Day 30 Day 40</td>
<td></td>
<td>Mean ± Sd Mean ± Sd Mean ± Sd Mean ± Sd Mean ± Sd</td>
<td></td>
</tr>
<tr>
<td>1*</td>
<td>12 M&amp;F</td>
<td>157.6±19.0 158.0±19.2 156.0±21.3 154.8±21.9 160.8±5.7</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>2*</td>
<td>12 M&amp;F</td>
<td>168.7±28.0 160.0±28.2 163.5±28.1 163.1±28.9 165.2±28.9</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3*</td>
<td>12 M&amp;F</td>
<td>152.16±8.0 152.8±7.3 153.7±6.4 152.9±12.7 188.5±7.7</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1* = Indicate 1g Jaman seed (powder) fed to the diabetic individuals.
2* = Indicate 3g Jaman seed (powder) fed to the diabetic individuals.
3* = Indicate 6g Jaman seed (powder) fed to the diabetic individuals.
Appendix-III: Effect of Jaman fruit on blood TGL in diabetic individuals (excluding the sex factor).

<table>
<thead>
<tr>
<th>Doses groups</th>
<th>Sex</th>
<th>Fasting Blood Glucose mg/dl</th>
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<tbody>
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<td></td>
<td>Before Intervention</td>
</tr>
<tr>
<td></td>
<td>Day 0</td>
<td>Day 10</td>
</tr>
<tr>
<td></td>
<td>Mean ± Sd</td>
<td>Mean ± Sd</td>
</tr>
<tr>
<td>1*</td>
<td>12</td>
<td>M&amp;F</td>
</tr>
<tr>
<td>2*</td>
<td>12</td>
<td>M&amp;F</td>
</tr>
<tr>
<td>3*</td>
<td>12</td>
<td>M&amp;F</td>
</tr>
</tbody>
</table>

1* = Indicate 6 Nos. of Jaman fruit fed to the diabetic individuals.
2* = Indicate 12 Nos. of Jaman fruit fed to the diabetic individuals.
3* = Indicate 18 Nos. of Jaman fruit fed to the diabetic individuals.
Appendix-IV: Effect of Jaman seed on blood TGL in diabetic individuals (excluding the sex factor).

<table>
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<th>Groups of Diabetics</th>
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<th>Fasting Blood Glucose mg/dl</th>
</tr>
</thead>
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<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td>Day 0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mean ± Sd</td>
</tr>
<tr>
<td>1* M&amp;F</td>
<td>12</td>
<td>227.8±26.9</td>
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<tr>
<td>2* M&amp;F</td>
<td>12</td>
<td>229.9±19.1</td>
</tr>
<tr>
<td>3* M&amp;F</td>
<td>12</td>
<td>236.3±22.7</td>
</tr>
</tbody>
</table>

1* = Indicate 1g Jaman seed (powder) fed to the diabetic individuals.
2* = Indicate 3g Jaman seed (powder) fed to the diabetic individuals.
3* = Indicate 6g Jaman seed (powder) fed to the diabetic individuals.
Appendix-V: Effect of Jaman fruit on blood Cholesterol in diabetic individuals (excluding the sex factor).

<table>
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<td></td>
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<tr>
<td></td>
<td></td>
<td>Day 0</td>
</tr>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>1*</td>
<td>12 M&amp;E</td>
<td>225.6±31.1</td>
</tr>
<tr>
<td>2*</td>
<td>12 M&amp;E</td>
<td>207.4±25.1</td>
</tr>
<tr>
<td>3*</td>
<td>12 M&amp;E</td>
<td>208.2±18.0b</td>
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</tbody>
</table>

1* = Indicate 6 Nos. of Jaman fruit fed to the diabetic individuals.
2* = Indicate 12 Nos. of Jaman fruit fed to the diabetic individuals.
3* = Indicate 18 Nos. of Jaman fruit fed to the diabetic individuals.
Appendix VI: Effect of Jaman seed on blood cholesterol in diabetic individuals (excluding the sex factor).

<table>
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<tr>
<th>Groups of Diabetics</th>
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<th>Fasting Blood Glucose mg/dl</th>
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<tbody>
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<tr>
<td></td>
<td></td>
<td>Day 0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mean ± Sd</td>
</tr>
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<td>M&amp;F</td>
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<tr>
<td></td>
<td></td>
<td>205.3±7.9</td>
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<td>2*</td>
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<td>M&amp;F</td>
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<tr>
<td></td>
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<td>197.3±35.0</td>
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<td>3*</td>
<td>12</td>
<td>M&amp;F</td>
</tr>
<tr>
<td></td>
<td></td>
<td>213.0±26.3</td>
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</tbody>
</table>

1* = Indicate 1g Jaman seed (powder) fed to the diabetic individuals.
2* = Indicate 3g Jaman seed (powder) fed to the diabetic individuals.
3* = Indicate 6g Jaman seed (powder) fed to the diabetic individuals.
**Appendix-VII:** Effect of Jaman fruit on HDL in diabetic individuals (excluding sex factor).

<table>
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<th>Doses groups</th>
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<td></td>
<td>Mean ± Sd</td>
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<td>12 M&amp;F</td>
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<td>3*</td>
<td>12 M&amp;F</td>
<td>36.8±12.7</td>
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1* = Indicate 6 Nos. of Jaman fruit fed to the diabetic individuals.

2* = Indicate 12 Nos. of Jaman fruit fed to the diabetic individuals.

3* = Indicate 18 Nos. of Jaman fruit fed to the diabetic individuals.
### Appendix-VIII: Effect of Jaman seed on blood HDL in diabetic individuals (excluding sex factor).

<table>
<thead>
<tr>
<th>Groups of Diabetics</th>
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<tr>
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<td>Mean ± Sd</td>
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<tr>
<td>1*</td>
<td>12 M&amp;F</td>
<td>35.0±6.6</td>
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<td>2*</td>
<td>12 M&amp;F</td>
<td>34.4±4.2</td>
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<td>3*</td>
<td>12 M&amp;F</td>
<td>27.7±2.2</td>
</tr>
</tbody>
</table>

- **1* =** Indicate 1g Jaman seed (powder) fed to the diabetic individuals.
- **2* =** Indicate 3g Jaman seed (powder) fed to the diabetic individuals.
- **3* =** Indicate 6g Jaman seed (powder) fed to the diabetic individuals.
Appendix-IX: Effect of Jaman fruit on blood LDL in diabetic individuals (excluding the sex factor).

<table>
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<tr>
<th>Doses groups</th>
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<td>Day 0</td>
</tr>
<tr>
<td></td>
<td>Mean ± Sd</td>
<td>Mean ± Sd</td>
</tr>
<tr>
<td>1*</td>
<td>12 M &amp; F</td>
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<td>2*</td>
<td>12 M &amp; F</td>
<td>136.5±17.9</td>
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<tr>
<td>3*</td>
<td>12 M &amp; F</td>
<td>147.7±28.6</td>
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1* = Indicate 6 Nos. of Jaman fruit fed to the diabetic individuals.
2* = Indicate 12 Nos. of Jaman fruit fed to the diabetic individuals.
3* = Indicate 18 Nos. of Jaman fruit fed to the diabetic individuals.
Appendix-X:  Effect of Jaman seed on blood LDL in diabetic individuals (excluding the sex factor).

<table>
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<th>Groups of Diabetics</th>
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<td>Before Intervention</td>
<td>During Intervention</td>
<td>After Intervention</td>
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</tr>
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<td></td>
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<td>Day 10</td>
<td>Day 20</td>
<td>Day 30</td>
</tr>
<tr>
<td></td>
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<td>Mean ± Sd</td>
<td>Mean ± Sd</td>
<td>Mean ± Sd</td>
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</tr>
<tr>
<td>1*</td>
<td>12 M&amp;F</td>
<td>157.6±19.0</td>
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<tr>
<td>2*</td>
<td>12 M&amp;F</td>
<td>168.7±28.0</td>
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<td>163.1±28.9</td>
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<tr>
<td>3*</td>
<td>12 M&amp;F</td>
<td>152.10±8.0</td>
<td>152.8±7.3</td>
<td>153.7±6.4</td>
<td>152.9±12.7</td>
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</table>

1* = Indicate 1g Jaman seed (powder) fed to the diabetic individuals.

2* = Indicate 3g Jaman seed (powder) fed to the diabetic individuals.

3* = Indicate 6g Jaman seed (powder) fed to the diabetic individuals.
Appendix-XI: Exchanges of Jaman Fruits

1 fruit exchange = 15 g carbohydrates, 0 g proteins & 0 g fats, 60 kcal

1. 6 fruits of Jaman = 48 g
   Nos. of exchanges in 6 fruits = 3.2 exchanges
   3.2 exchanges = 48 g
   1 exchange = 15 g of jaman fruits

2. 12 fruits of Jaman = 96 g
   Nos. of exchanges in 12 fruits = 6.4 exchanges

3. 18 fruits of Jaman = 96 g
   Nos. of exchanges in 12 fruits = 9.6 exchanges

Appendix XII.  QUESTIONNAIRE FOR SCREENING TEST

1. Name------------------  2. Address----------------

3. Occupation------------------  4. Sex----------------

5. Age------------------  6. Exercise  yes--No---

If yes specify------------------

7. Medication for diabetes (Name of Medication)------------------(NO INSULIN).

8. Dose M -------------A -------------E ----------------

9. Any medication for other conditions. Yes ------------ No--

10. Are you using sweets sugars. Yes ------------ No ------------

11. Glucose at the screening test ------------ mg/dl/

12. Triglyceride at the screening test ------------ mg/dl
Appendix XIII

Consent form

اس بات کا اقرار کرتا ہوں کہ کوئی بھی نہیں ہے جس کی مشکلات ہوں گیا ہے۔

دیکھو نے دویں اس حالت سے کامیاب ہوگا جب کوئی بھی اس کا مزاحیہ سے کسی میں خجالت نہیں ہے۔

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